THE PULMONARY FUNCTION TEST IN TYPE-2 DIABETICS AND NON-DIABETICS-A COMPARATIVE STUDY

DISSERTATION SUBMITTED FOR

M.D GENERAL MEDICINE

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MEDICAL UNIVERSITY CHENNAI,

TAMILNADU,INDIA

CERTIFICATE FROM THE DEAN

This is to certify that the dissertation entitled **"PULMONARY FUNCTION TEST IN TYPE-2 DIABETICS AND NON-DIABETICS-A COMPARATIVE STUDY"** is the bonafide work of **Dr. ILAMARAN.M**, in partial fulfillment of the university regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for M.D General Medicine Branch I examination to be held in April 2016.

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DECLARATION

I, **Dr.ILAMARAN.M**, solemnly declare that, this dissertation **"PULMONARY FUNCTION TEST IN TYPE-2 DIABETICS AND NON-DIABETICS-A COMPARATIVE STUDY"** is a bonafide record of work done by me at the Department of General Medicine, Kanyakumari government medical college hospital, Asaripallam, under the guidance of **Dr. M. CHRISTOPHER NESAMONY, M.D**, Professor, Department of General Medicine, Kanyakumari Govt Medical college, Asaripallam. This dissertation is submitted to The Tamil Nadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulations for the award of M.D Degree General Medicine Branch-I;

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CONTENTS

INTRODUCTION

The management of endocrine disorders requires the understanding of intermediary metabolism, reproductive physiology, bone metabolism, and growth. The practice of endocrinology is intimately linked to conceptional framework for understanding hormone secretion, hormone action and principles of feedback control.

Thyroid hormone controls about 25% of basal metabolism of most of the body tissues, cortisol exerts a permissive action for many hormones in addition to its own direct effects, PTH regulates calcium and phosphorus levels, vasopressin regulates serum osmolality by controlling water balance, mineralocorticoids control minerals metabolism, insulin maintains euglycemia in the fed and fasted states.

The causes of endocrine dysfunctions are mainly due to

1.Hyperfunction:

-neoplastic

-autoimmune

-iatrogenic

-infectious

-inflammatory

- activating receptor mutation.

2.Hypofunction:

-autoimmune

-iatrogenic

-hormone mutations

-enzyme effects

-develepmental defects

-nutritional or vitamin deficiency

-hemorrhage or infarction

3. Hormone resistance:

-receptor mutation

-signalling mutation

-post receptor mutation

Among all the endocrine conditions obesity is the highly prevalent one occupies about > 30% followed by hyperlipidemia, type 2 diabetes mellitus, hypothyroidism, erectile dysfunction, hirsuitism, gynaecomastia, vitamin deficiencies.

Type 2 diabetes mellitus is nothing but a persistent hyperglycemia and altered metabolism of lipids, carbohydrates and proteins. Several distinct types of diabetes mellitus are caused by complex interactions between genetics and environmental factors. These are result from impaired insulin secretion and insulin resistance or combination of both of these mechanisms.

Type 2 diabetes mellitus is associated with chronic tissue damage, reduction in function, failure of multiple organs and its complications are preferably caused by macrovascular and microvascular damages and all are due to the metabolic dysregulation of the diabetes mellitus.

Though great attention was centered on the diabetic complications which had cardiovascular nature, nephropathy, retinopathy and neuropathy, the pulmonary complications of type2 diabetes mellitus have been poorly characterized. Of late the concept of the lung as a target organ for diabetic microangiopathy is receiving continuing attention. The aim of the study was to assess the effects of chronic hyperglycemia on lung functions, which focused on mechanical aspects of lung dysfunction, maximal forced spirometric pulmonary function tests like FVC, FEV1, FEV1/FVC to be specific[1].

Spirometry is the pulmonary function tests which read the mechanical lung function, which measures air that can be inhaled and exhaled.

The pulmonary complications of type 2 diabetes mellitus have been poorly characterized[1, 2] .The complications are affect the lungs silently and may produce increased morbidity because of lung dysfunction[2].

Relatively few studies have been done on pulmonary mechanical function. Our study mainly concentrating on mechanical dysfunction of lungs due to diabetes mellitus mainly maximal forced spirometric PFTs to be specific. Most of the studies were done on type1diabetics[3]. The present study was done on type 2 diabetics.

Because chronic disease affecting pulmonary function tests are having only few kind of studies only. But chronic endocrine disorder like in our study the type 2 diabetes mellitus also affects the lung. For all pulmonary complications the mechanism of damage is chronic hyperglycemia. So many lung disorders have been noted in patient with type 2 diabetes mellitus of which we are going to see mainly the mechanical complication of lung due to diabetes.

REVIEW OF LITERATURE

DIABETES MELLITUS:

Diabetes mellitus is characterized by chronic hyperglycemia due to defects in insulin secretion, peripheral insulin action or both which leading to alteration in the fat, proteins and carbohydrate metabolism of the individual[5].

Type 2 diabetes affects all types of ethnicity, social and economic levels of the society. Diabetes is among the 5 leading cause of death in most countries. Better and early disease detection, changing life styles and changes in the diagnostic criteria have led to this increase.

Death and disability associated with diabetes poses a serious challenge to physicians and the health system at large.

ETIOLOGIC CLASSIFICATION OF DIABETES:

1) Type 1 diabetes (Beta cell destruction, leading to absolute insulin deficiency)[6]

    a) Immune mediated

    b) Idiopathic

2) Type 2 diabetes – range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance[6]

3) Other specific types[6]

a) Genetic defects of beta cell function

1. Maturity onset diabetes of the young 1-6
2. Mitochondrial DNA
3. Mutant insulins
4. Hyperinsulinemia
5. Others

b) Genetic defects in insulin action

1. Type A insulin resistance
2. Leprechaunism
3. Rabson-Mendenhall syndrome
4. Lipoatropic diabetes
5. Others

c) Diseases of exocrine pancreas

1. Pancreatitis
2. Trauma / Pancreatectomy
3. Neoplasia

4. Cystic fibrosis

5. Hemochromatosis

6. Fibro-calculous pancreatopathy

7. Others

d) Endocrinopathies

e) Drug or chemical induced

1. Vacor

2. Pentamidine

3. Nicotinic acid

4. Glucocorticoids

5. Thyroid hormone

6. Diazoxide

7. Beta adrenergic agonists

8. Thiazides

9. Phenytoin

10. Alpha interferon

11. Protease inhibitors

12. Atypical antipsychotics

f) Infections

1. Congenital rubella

2. Cytomegalovirus

3. Others

g) Uncommon forms of immune mediated diabetes

1. “Stiff man syndrome”

2. Anti-insulin receptor antibodies

3. Others

h) Other genetic syndromes associated with diabetes

1. Down’s syndrome

2. Klinefilter’s syndrome

3. Wolfram’s syndrome

4. Friederich’s ataxia

5. Huntington’s chorea

6. Laurence Moon Biedel syndrome

7. Myotonic dystrophy

8. Porphyria

9. Prader willi syndrome

10. Others

4) Gestational diabetes mellitus[5, 6]

CRITERIA FOR DIAGNOSIS OF DIABETES MELLITUS:[5, 6, 7]

1. Symptoms of diabetes with casual random blood glucose – 200 mg/dl (11.1 mmol/L)

Random is defined as any time of delay without time of the last meal.

The symptoms of diabetes are polyuria, polydipsia, and unexplained weight loss

OR

2. Fasting blood glucose > 126 mg/dl (7.0 mmol/L)

Fasting is defined as no caloric intake for atleast 8 hour

OR

A1C > 6.5%

OR

3. 2- hr postprandial blood glucose >200 mg/dl (11.1 mmol/L) during an OGTT. The test should be performed as described by WHO, using a glucose load containing the equivalent of 75g of anhydrous glucose dissolved in water.

SCREENING OF DIABETES MELLITUS:

Widespread use of fasting plasma glucose and A1C as a screening for diabetes mellitus is recommended because

1. A large number of individual who met the criteria foe diagnosis are mostly asymptomatic only and unaware of that they having the disorder.
2. Epidemiologic studies suggested that type 2 diabetes mellitus may be present for up to the decade before diagnosis.
3. Some people having one or more diabetic complication at the time of diagnosis itself.
4. The treatment of diabetes mellitus may alter the natural history of the disease.

ADA recommends screening of all individuals of more than 45 yrs of age and screen in the earlier incase of having overweight, overweight defined as the body mass index of more than 25 kilogram/ meter square[8].

RISK FACTORS FOR TYPE 2 DIABETES MELLITUS:

1) History of diabetes mellitus in family members
2) Obese (Body mass index > 25/meter square)[9]
3) Sedantary life [9]
4) Past history of Impaired fasting glucose or Impaired glucose tolerance or and A1c of 5.7 – 6.4% [9]
5) Past History of gestational diabetes mellitus or delivery of a baby of > 4 kilograms [9]
6) High density cholesterol < 35 milligrams/dl (0.90 mmol/L) and /or a triglyceride > 250milligrams/dl (2.82mmol/L) [9]
7) Polycystic ovarian disease [8, 9]
8) History of vascular disease [9]
9) History of cardiac disease [8, 9]
10) Hypertension of more than 140/90 mmhg [9]

PATHOGENESIS FOR TYPE 2 DIABETES MELLITUS:

Earliest abnormality seen in type 2 diabetes is impairment in tissue sensitivity to insulin. This results in an increase in demand on the beta cell to maintain a sufficiently high rate of insulin secretion to the offset of insulin resistance. After a certain time, when the insulin secretion fails to meet the insulin demand, overt diabetes occurs.

There is a very clear understanding for the role of genetic factors in development of type 1 diabetes however; it is not the case with type 2 diabetes. Type 2 diabetes is caused by interactions between the environmental factors and genetic factors.

DEFECTIVE 1ST PHASE INSULIN SECRETION:

This phase of insulin secretion helps in priming the insulin target tissues to maintain the normal glucose homeostasis. It is one of the early manifestations, which is found to occur when fasting glucose rise to 115-120mg% [10]

DEFECTIVE PULSATILE INSULIN SECRETION:

Insulin is normally secreted in pulses of rapid frequency [10]. Abnormal oscillatory insulin secretion is the characteristic of earlier part of type 2 diabetes.

FACTORS AFFECTING BETA CELL FUNCTION:

- Chemical toxins like alcohol
- Malnutrition
- Chronic pancreatitis
- Amylin accumulation
- Intrauterine environment
- Drugs like thiazides, beta blockers etc…
- Glucose transporter 2 defect
- Decreased glucokinase activity
- Defects in phosphoinositol

ROLE OF LIVER IN GLUCOSE METABOLISM:

1. Liver is the organ of glucose production and glucose consumption
2. It is exposed to insulin concentration in the portal circulation which is 3 – 10 times more the systemic circulation[11].
3. Sole site of glycol regulatory action of glucose[11, 12].
4. Absorbed hexose's reach liver before reaching muscle and adipose tissues.

Liver has storage of glucose and glycogen about 70gm at a time. 75% of hepatic glucose output comes from gluconeogenesis[11, 12].

Contribution of liver in glucose homeostasis depends on the following factors:

1. Sensitivity of hepatocytes to small increments in insulin levels
2. Ratio of insulin to glucagon
3. Responsiveness of glycogenolysis and gluconeogenesis to hormonal modulation[11].

PRESENTATION OF DIABETES MELLITUS:

Diabetes can be detected in one of the following ways. Some patients are found to have excess of glucose or sugar in urine incidentally on routine checkup without any complaints or physical signs.

Some patients are found to have diabetes while investigating for an associated complaint like, ischemic heart disease, hypertension, eye diseases, kidney disease, non- healing foot ulcers etc[12].

Diabetes can manifest as an acute illness – ketoacidosis with an acute infection or even without evidence of any cause. Pain abdomen and vomiting may be the presenting complaints in some patients. This most commonly occurs in juvenile onset diabetes mellitus.

Some of the patients often present with classical symptoms of diabetes. Eg., excessive thirst, frequent micturition, increased appetite, weight loss, severe weakness, repeated infections, itching in genitals, diminished vision, numbness in limbs and occasionally impotence[13].

COMPLICATIONS OF DIABETES MELLITUS:

DIABETIC METABOLIC EMERGENCIES:

The two main metabolic complications of diabetes are diabetic ketoacidosis and hyperosmolar state[12]. Initially DKA was considered as the main complication of Type 1 diabetes mellitus. Both are associated with relative insulin deficiency, volume depletion and acid base abnormalities.

PATHOPHYSIOLOGY:

1. Non-enzymatic glycosylation of proteins, e.g. Hemoglobin, collagen, LDL and tubulin in peripheral nerves[14].
2. Polyol pathway[13, 14].
3. Abnormal microvascular blood flow to the peripheries[14].
4. Reactive oxygen species and growth factors stimulation (TGF -J3) and vascular endothelial growth factor (VEGF) [14].
5. Blood hemodynamic changes.

MACROVASCULAR COMPLICATIONS:

Diabetes is a risk for the development of atheroma.

This includes

a) Ischemic heart disease

b) Peripheral vascular disease

c) Cerebrovascular accidents

The cardiovascular complications are more in diabetes mellitus. So the patient with diabetes mellitus with suspected cardiovascular risk factors has to be started with an anti-hypertensive mainly ACE inhibitor, a statin and a low dose aspirin for all the patients[15] unless otherwise contraindicated.

MICROVASCULAR COMPLICATIONS:

Small blood vessels all over the body are affected but the disease process is of danger in 3 sites:

a) Eye – Retionopathy

b) Neuropathy –(mono and polyneuropathy)

c) Nephropathy

DIABETIC RETINOPATHY:

Diabetic retinopathy is divided into proliferative diabetic retinopathy (PDR) and non-proliferative diabetic retinopathy (NPDR). Hemorrhages or micro aneurysms, cotton wool spots, hard exudates, intra retinal microvascular abnormalities, venous caliber abnormalities like venous loops, venous bleeding and venous tortuosity are some of the findings associated with early and progressive diabetic retinopathy. Micro aneurysms and saccular out-pouchings of the capillary walls can leak fluid and results in intra edema and hemorrhages[16]. These intra retinal hemorrhages are flame shaped or dot blot like appearance, reflecting the architecture of the layer of the retina in which they occur. Flame shaped hemorrhages occur in inner retina closer to the vitreous and dot blot hemorrhages occur deep in the retina. Intra retinal microvascular abnormalities are either new vessel growth within retinal tissue or shunt vessels through areas of poor vascular perfusion.

The micro infarcts in the nerve fibre layer of retina are called cotton wool spots. Retinal detachment can occur due to neovascularization with fibrous tissue contraction that can distort contraction that can distort retina and lead to traction.

DIABETIC NEUROPATHY:

Diabetic neuropathy has a number of clinical syndromes with subclinical or clinical manifestations depending upon the class of nerve fibres involved, it can manifest as polyneuropathy, mono neuropathy or autonomic neuropathy.

Distal symmetrical polyneuropathy is the most common form of diabetic neuropathy, where patients frequently present with distal loss of sensation, hyperesthesia, paresthesia and dysthesia. Symptoms may include a sensation of numbness, tingling, sharpness that begins in feet and spreads proximally. As the diabetes progresses, the pain subsides and eventually disappears, but a sensory deficit in lower limbs persist.

Diabetic poly-radiculopathy is a syndrome characterized by severe disabling pain in the distribution of one or more nerve roots, which can be accompanied by motor weakness. There can be severe pain in hip and thigh due to involvement of lumbar plexus. Fortunately this condition is usually self-limited and resolves in 6 -12 minutes.

Mononeuropathy is less common in diabetes which presents with pain sensation and weakness in the course of a single nerve. Third cranial nerve is more commonly involved and heralded by diplopia. Sometimes 4^{th},6^{th} and 7^{th} cranial nerves are also involved[15]. Peripheral mono neuropathies can also occur.

Autonomic neuropathy in diabetes can involve multiple systems like cardiovascular, gastrointestinal, genito-urinary[17] etc. patient can have resting tachycardia and orthostatic hypotension when cardiovascular system is involved. Gastropathy and bladder dysfunction are caused by autonomic neuropathy of gastrointestinal tract and genitourinary tract.

Increased sweating of upper extremities and decreased sweating of lower extremities can occur due to sympathetic nervous system dysfunction. There is a increased chance of foot ulcers due to anhidrosis of lower extremities which causes dry skin and cracking.

Autonomic neuropathy can produce hypoglycemic unawareness, that means it decreases the counter regulatory hormones of glucose metabolism in the period of hypoglycemia. So the hypoglycemia episodes are more common with diabetic neuropathy.

The treat for diabetic neuropathy is less than satisfactory only. The blood sugar controlling is the mainstay of treatment. The treatment options are anti depressents, anti epileptics. Now the newer drugs are pragabalin and the duloxetine.

NEPHROPATHY:

Nephropathy secondary to diabetic glomerulopathy occurs after diagnosis is about 15-20years and 25-35% of patients detected under the age of 30 years.

It may the leading cause of premature death in diabetic patients in the young individuals.

Diabetic nephropathy characterized by hypertension, proteinuria and renal impairment.

The diabetes affects kidney by:

- Glomerular insult
- Ischemia from hypertrophy of afferent and efferent arterioles
- Ascending mode of infection.

The earliest abnormality of the diabetic kidney has the renal hypertrophy associated with an increased glomerular filtration rate. This appears immediately after diagnosis and is related to poor diabetic control.

Diabetic nephropathy has 5 stages based upon the glomerular filtration rate, of which 1 -3 are considered as pre-clinical or “silent” and stages 4 and 5 are called as clinical diabetic nephropathy.

Stage	Description	GFR (mL/min/1.73 m^2)
1.	Kidney damage with normal or elevated	Glomerular filtration rate >90
2.	Kidney damage with mildly decreased	Glomerular filtration rate 60–89
3.	Moderately decreased	Glomerular filtration rate 30–59
4.	Severely decreased	Glomerular filtration rate 15–29
5.	Kidney failure	<less than 15 (or dialysis)

Stage 1:

Here glomerular filtration rate is elevated on an average by 20-40% above that of age matched normal controls in both adults and children with type 1 DM resulting in glomerular enlargement.

Stage 2 :

It is an extension of stage 1. Histopathological changes like basal membrane thickness and mesangial expression begins to be detected within 1st two years after the onset of IDDM. This stage is usually silent clinically with

normal albumin excretion rate despite structural changes. Most patients remain in stage 2, however 30-40% progress to subsequent stages.

Stage 3:

Here patients will have microalbuminuria detected. Patients who have albumin excretion rates >30 microgm/min are more likely to develop clinical diabetic nephropathy than those with less than 30 microgm/min.

Stage 4:

In this stage, patients develop overt nephropathy. By definition, patients have persistent clinical proteinuria with albumin excretion rate of > 250 microgm/min in 24 hrs, hypertension and a decrease in GFR. Once proteinuria is persistently present, development of end stage renal disease or death occurs in 3 – 4.8 yrs.

Stage 5:

This is a stage of advanced renal failure. In contrast to non-diabetic patients, diabetics with end stage renal disease usually have other systemic manifestations in addition to their renal diseases.

The earliest manifestation of nephropathy is microalbuminuria which means it does not detected by normal dipstick method. The massive proteinuria

can induce a transient nephrotic syndrome, anasarca and decreased albumin level.

Patients with nephropathy show in peripheral smear normochromic normocytic anemia and an elevated erythrocyte sedimentation rate (ESR).

Development of hypertension may itself damage the kidney still further.

Effective treatment of blood pressure in a target of less than 130/80 mmHg has been shown to delays the progression of renal failure considerably.

ACE inhibitors or angiotensin receptor blockers are the drugs of choice.

For evaluating diabetic nephropathy initially have to do microalbuminuria test as a spot collection and excludes the conditions that causes the increase albumin excretion, and repeat it every 3-6 months period. If two of the three microalbuminuria test have positive results then we have to start the treatment with either ACE inhibitors or angiotensin receptor blockers.

The early starting of the treatment for nephropathy is must because if it prolongs it will end up in the chronic kidney disease. Because survival after attaining the end stage renal disease is very shorter and the patients may ends with renal replacement therapy and also had increasred risk of developing the dialysis related complications.

PULMONARY COMPLICATIONS:

Major consequences of hyperglycemia are excessive non enzymatic glycosylation of various body proteins including albumin[1, 3, 7], collagen and elastin. In type 2 diabetics with uncontrolled disease there is decreased pulmonary functions have been noted. There may be increased cross linkage between polypeptides of collagen which leading on to thickening leading to restriction of lung volume and alveolar gas transport, reduced membrane diffusion capacity and pulmonary capillary blood volume[15,16, 17]. The possible explanations of restrictive type of lung disease are thickening of alveolar epithelium, pulmonary microangiopathy and centrilobular emphysema. So the net effect due to collagen & elastin changes and microangiopathy[1`, 2 , 18, 19,.

The lung complications of type 2 diabetes mellitus are mainly affects the

Mechanical aspects of the organ and among the that the restrictive pattern of lung disease is the commonest one, these effects are reported by using the spirometry and measuring the forced vital capacity and forced expiratory volume in first second and ratio between these two.

OTHER COMPLICATIONS:

a) Cardio vascular

b) Gastrointestinal tract (decreased motility, diarrhea)

c) Genitourinary tract (urological abnormalities, impotence)

d) Infections

e) dermatological

f) glaucoma

g) cataracts

h) periodontal diseases

i) hearing loss

j) lower extremity complications

k) Dyslipidemia

l) Hypertension

METABOLIC SYNDROME:

Insulin resistance and hyper insulinemia is being increasingly implicated in the pathogenesis of various metabolic disturbances. Some of them has been clubbed under a syndrome.

Diagnosis of metabolic syndrome is made in the presence of atleast three of the following –

1. Waist circumference >102 centimeter in male and >88 centimeter in females

2. Triglycerides >150mg/deci litre or patients using nicotinic acid or fibrates

3. HDL <40mg/deci litre in males and <50mg/deci liter in females

4. Systolic Blood pressure >130mmHg or diastolic Blood pressure >85 mmHg or patients using anti hypertensive drugs

5. Fasting blood sugar > 100mg/dl or patients using OHA's.

The main mechanism of the metabolic syndrome is the hyperinsulinism that causes the various properties of the disease and cause syndrome X manifestating as hyperinsulinism, hypertension and obesity[7, 8].

CHILDREN, ADOLESCENTS,AND YOUNG ADULTS:

Diabetes in children and teenagers is generally ketosis prone, insulin deficient type 1 diabetes, but cases of type 2 diabetes in obese children are now occurring and the possibility of a diagnosis of maturity onset diabetes of the young should be considered, especially when there is a positive family history of diabetes. Adherence to the treatment is tough in adolescents, so the deterioration of glycemic control is common[18].

Wide fluctuations of blood glucose and frequent metabolic emergencies are sometimes observed in adolescents and young adults but the use of the term brittle diabetes is discouraged, as this is not considered to be a pathological entity[18, 20]. Studies have shown that this problem is associated with persistent manipulation of therapy to induce recurrent diabetic ketoacidosis or severe hypoglycemia requiring admission. This attention seeking behavior may be manifestation of psychological disturbance, is factitious and is not a specific phenomenon peculiar to some aspect of diabetes or its management in susceptible individuals.

MANAGEMENT OF TYPE 2 DIABETES:

LIFESTYLE INTERVENTION:

Reducing weight and physical exercise are also the important part in the management of diabetes mellitus. Increase in weight with diabetes mellitus and decreased physical activity increases the complications of diabetes mellitus and increases the mortality.

Proper exercise and diet control are the key points in the management of diabetes mellitus. But the long term complications are which are not controlled by exercise and diet control alone, it needs immediate drug management.

Along with improved drug compliance the patient education allows individual with diabetes mellitus having the responsibility for their care. Patient education as continued visits to the hospitals for reinforcement. The ADA refers to education about the individualized management plan for the patient as diabetes self management education, for more frequent contact between the patient and the diabetes management team that improves the glycemic control of the patient with diabetes mellitus.

FOOD SELECTION:

RECOMMENDED COMPOSITION OF DIET:

Carbohydrate- 45-60% daily

Sucrose- up to 10%

Fat (total) <35%

n-6 polyunsaturated- <10%

n-3 polyunsaturated- eat oily fish once or twice weekly

monounsaturated- 10-20%

saturated- <10%

protein- 10-20% (do not exceed 1gram per kilogram body weight)

Plate model may provide a simple visual aid to show the proportions of carbohydrates and other food groups for selection at mealtimes.

According to the medical nutritional therapy the nutritional recommendations for diabetic patients are hypocaloric diet having low fat or low carbohydrate diet, minimal trans fat consumption, protein in diet as a part of optimal diet, non-nutrient sweeteners, and the routine suppliments of vitamins, antioxidants, the trace elements are not advised[17,20].

PHYSICAL ACTIVITY:

Increase in the physical activity among the people with diabetes mellitus is the first main treatment in the management of diabetes mellitus. Aerobic exercise is advisable. The importance of exercise is to reduce the weight and it increases the insulin sensitivity in the target organs by increasing the receptors for the activity.

The physical activity is individualized according to the patients performance status and comorbidities and the age and the risk factors. In the patient with diabetes mellitus ADA recommends about 150minutes per week of exercise that is distributed over at least 3days, of moderate aerobic exercise[20, 21]. The exercise regimens also includes the resistance training.

The one of the major complication is exercise induced hypoglycemia that too more common in type 1 diabetic patients and can occur with patient on insulin or insulin analogues.

Because the asymptomatic cardiovascular manifestations are occur in the younger age in patients with type 1 and 2 diabetics. So the formal exercise testing is warranted in patients with age>35 years, diabetes duration of more than 15years, with microvascular complications of diabetes mellitus, autonomic neuropathy and other risk factors for coronary heart disease[21].

MEDICATIONS:

The goal of therapy in type 2 diabetes should not only improve the beta cell function, but also enhance the glucose utilization in the peripheral tissues. These oral drugs also have the potential to correct the hormonal and metabolic abnormalities in diabetes.

SULFONYLUREAS:

Sulfonylureas act by binding to the so called sulfonylurea receptors on the pancreatic beta cell membranes causing depolarization, calcium influx and degranulation of secretory granules with insulin release. Therefore, these drugs primarily augment second phase of insulin secretion and has very less action on first phase.

Drugs – Tolbutamide, Chlorpropamide, Glimepride, Glipizide, Glibenclamide, Gliclazide.

The more rapid onset of action of sulfonylureas, lesser is the delay in the postprandial insulin release. Glipizide results in rapid post prandial insulin release and lowers post prandial glucose. In contrast glibenclamide exerts a better effect on fasting glucose. Hence, where fasting glucose levels are high, glibenclamide may be preferred to glipizide and vice versa.

Even among them, there are still reasons which are not clear for about 15 – 20% of patients who have no effect or little effect in glycemic control to

sulfonylurea therapy. Secondary failure, decresing the beta cell function and increasing the insulin resistance are important disease related factors for sulfonylurea failure besides the duration of the disease.

BIGUANIDES:

Metformin, phenformin and buformin belongs to this group. The later two drugs are withdrawn long back. Metformin is an old, but still the best agent of choice to start with in treatment of type 2 diabetes. Its efficacy, safety profile and its capacity to be associated with other anti-diabetic agents makes metformin the first line glucose lowering drug of choice in diabetes management.

The principle site of action is in the liver and muscles. The effort of metformin on liver is mediated by activation of liver kinase B1. This drug is prefered in normal weight patients, contrary to its widespread perception in our country that it is preferred in obese patients.

But the major complications of the patients with metformin on chronic may produce the vitamin B12 deficiency and lactic acidosis. So these drugs are mainly contraindicated in patients more prone for developing acidosis like chronic renal failure.

THIAZOLIDINEDIONES:

Thiazolidinediones are potent insulin sensitisers, that act through the PPAR gamma This PPAR gamma mediated transcriptional effects have are improves whole body sensitivity to insulin. Drugs used are Troglitazone, Rosiglitazone and pioglitazone. Among these drugs, troglitazone have been banned in the year 2000, due to its fatal hepatotoxicity after which rosiglitazone was introduced. Pioglitazone is the third drug used that has been shows to improvement of sensitivity of human body insulin.

GLP-1 ANALOGUES:

This incretin hormone GLP-1 is secreted from intestinal L cells, in the distal part of ileum and colon after food intake. The effect of subcutaneous injection of GLP-1 analogue is very short acting due to N terminal degradation by the enzyme dipeptidyl peptidase IV, restricting its cardiac use.

Drugs include Exenatide, Lexixinatide, Liraglutide, Exenatide LAR.. These drugs has beneficial effect on cardiovascular system lipid profile, obesity and also in central nervous system.

Now a days most of the physicians are using the liraglutide because it is a once daily dosage and apart from the sugar control it is also having the weight reducing property.

DIPEPTIDYL PEPTIDASE- IV INHIBITORS:

DPP-IV inhibitors are novel anti-diabetic drugs based on the incretin therapy. These drugs help in decreasing the degradation of endogenous incretin hormone. Gliptins are generally considered as weight neutral agents and may assist a small amount of weight loss. Drugs include – Sitagliptin, Vidagliptin, Saxagliptin, Linagliptin.

ALPHA – GLUCOSIDASE INHIBITORS:

Acarbose and Voglibose are these drugs. Mechanism of action of alpha glucosidase inhibitors involves block of the enzyme alpha glucosidase in the intestine which normally clears carbohydrates into absorbable monosaccharides.

ROLE OF INSULIN IN TYPE 2 DIABETES:

Type 2 diabetes constitutes nearly 95- 97% of all diabetics. The successful management of type 2 diabetes involves around an individual tailored nutritional plan, exercise regimen, use of oral agents and or insulin.

Tissues insensitivity to insulin and impaired insulin secretion are the pathogenetic factors underlying type 2 diabetes. The primary defect is tissue insensitivity to insulin.

DCCT provides strong evidence that tight glycemic control prevents microvascular complications. Hyperglycemia is a predictor of excessive cardiovascular mortality.

INDICATIONS FOR INSULIN USE IN TYPE 2 DIABETICS:

a) Primary oral agent failure

b) Secondary oral agent failure

c) Peri-operative

d) Pregnancy

e) Acute on chronic sepsis

f) Acute medical or surgical event

g) Major organ failure

h) Glucotoxicity

Hypoglycemia and weight gain are the 2 main complications of insulin therapy. Insulin is usually started as single dose of long acting given either before breakfast or just before the bed time . since the fasting hyperglycemia and increased hapatic production are the features of diabetes so that the bed time insulin is advisible now. Started as 0.3 tp 0.4 units per kilogram body weight.

WHEN TO START INSULIN?:

a) At diagnosis , fasting glucose > 200mg/dl ,post prandial glucose > 300 mg/dl with HbA1c >9%[22].

b) After oral; drug failure, despite receiving optimal dose of 2 or 3 OAD's, fasting glucose > 150 mg/dl, post prandial glucose > 200mg/dl with HbA1c levels of >8.5% [22] .

PROPERTIES OF INSULIN PREPARATIONS:

Type of preparation	Time onset (hours)	Peak level (hours)	Duration of action (hours)
Lispro	< 0.25	0.5-1.5	3-4
Aspart	<0.25	0.5-1.5	3-4
Glulisine	<0.25	0.5-1.5	3-4
Regular	0.5-1.0	2-3	4-6
Detemir	1-4		Up to 24hrs
Glargine	1-4		Up to 24hrs
NPH	1-4	6-10	10-16
75/25 – 75%protamine lispro,25% lispro	< 0.25	1.5	10-16

50/50- 50% protamine lispro,50lispro			
70/30-70%NPH,30% regular	0.5 -1	dual	10-16

Factors affecting the disposal of injected insulin:

1. Anatomic site
2. Exercise
3. Depth
4. Insulin concentration
5. Mixing of preparations
6. Local tissue degradation
7. Intra subject coefficient variation
8. Antibody binding and release of insulin
9. Physical state of modified insulin in serum [5,23]

GESTATIONAL DIABETES MELLITUS:

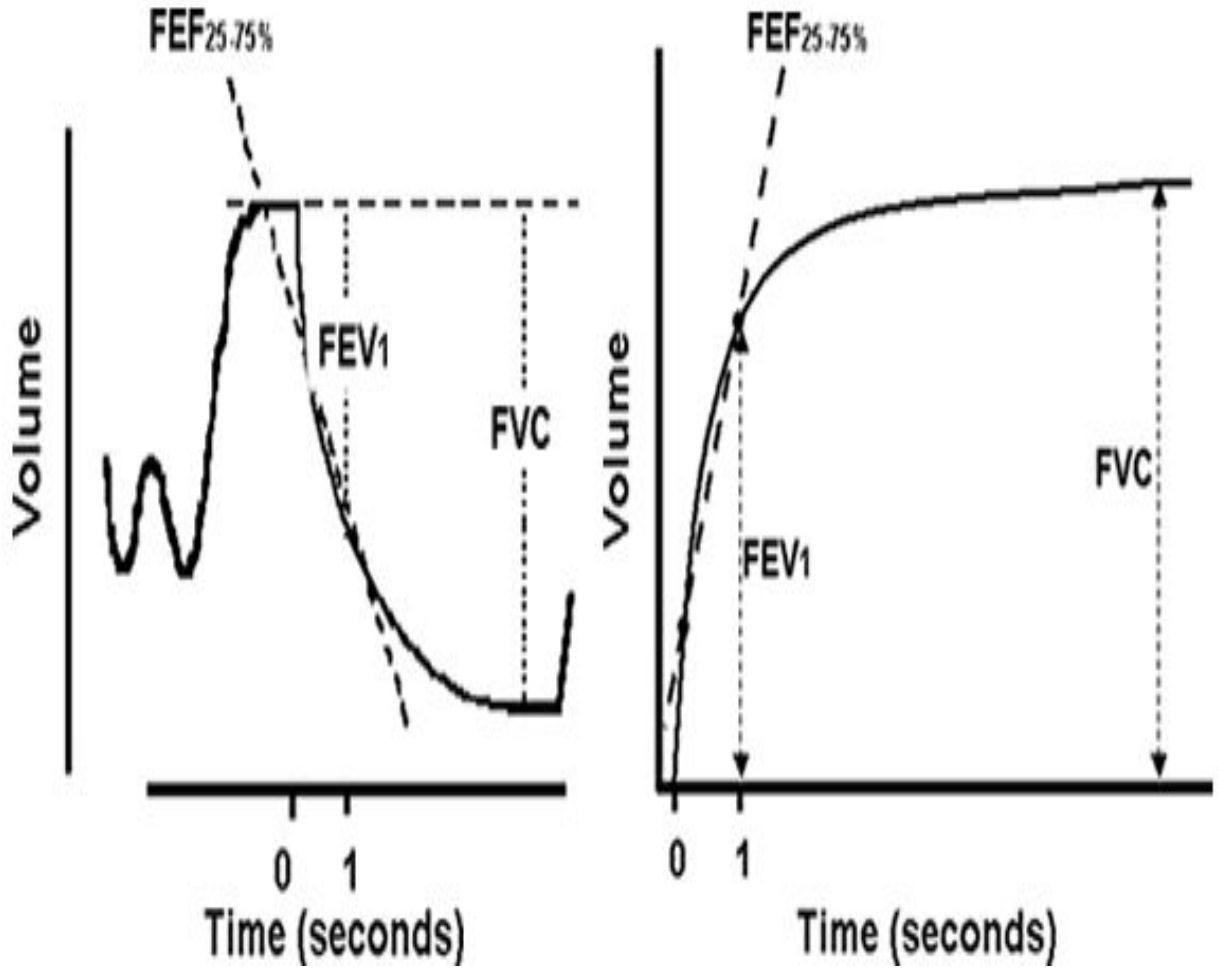
Glucose metabolism is altered during the normal pregnancy. There is a continuous relationship between maternal blood glucose and risk of adverse perinatal out comes . So that there is no threshold to defines the risk. According to WHO criteria all women meeting criteria for impaired glucose tolerance or diabetes after 75grams of oral glucose tolerance test having gestational diabetes. European criteria for diagnosis for gestational diabetes are a venous blood glucose of more than 99mg/dl in fasting state and of more than 162mg/dl of 2hr postprandial[24].

RISK FACTORS FOR GESTATIONAL DIABETES:

1. Obesity
2. Ethnicity (south Asian, black, Hispanic, native American)
3. Family history of type2 diabetes
4. Previous glucose abnormalities in pregnancy
5. Previous macrosomia

perinatal mortality rate is 3 to 4 times higher than those of the non-diabetic population, and the rate of congenital malformation is increased about 5to6 fold when comparing to normal pregnancy.

SPIROMETRY & PFT:



Spirometry is the recording of lung volumes and capacities by using spirometer. It is Simple, office-based, Measures flow, volumes, volume vs. Time. It is the most readily available most useful pulmonary function test .It takes 10 to 15 minutes and carries no risk. Spirometry is the most commonly used lung function screening study. It should be the clinician's first option. Other studies being reserved for specific indications. It is easily performed.

Used in the ambulatory setting, physician's office, emergency department, or inpatient setting [1,5,8,24].

It needs patients voluntary effort to inhale maximally beyond the tidal volume and exhale forcefully into the close circuit maneuver. Before doing the test the examiner must explain the complete procedure to reduce the faults[10].

Can determine:

- Forced expiratory volume in first second (FEV_1)
- Forced vital capacity (FVC)
- FEV_1/FVC
- Forced expiratory flow 25%-75% (FEF_{25-75})

Indications for Diagnosis:

1. Evaluate the patient having sudden onset of dyspnea, exertional dyspnea, chronic cough.
2. Screen the high risk populations.
3. Monitoring pulmonary toxicities of certain drugs.
4. Abnormalities in chest x ray, acid blood gas analysis and hemoglobin.
5. Preoperative evaluation.
6. Smokers more than 45 years (former & current) [27].

Indications for Prognosis:

1. Assess for severity of illness.
2. Therapy response.
3. Requirements of further treatment plan.
4. Surgery referral.
5. Disable

Contraindications for spirometer:

Relative contraindications for spirometer includes recent heart disease, unstable angina, abdominal aorta aneurysm, recent eye surgery, recent surgeries in the past, hemoptysis of unknown origin hemoptysis of unknown origin, pneumothorax, cerebral artery aneurysm, syncopal attacks, thoracic aorta aneurysm[28, 29].

Simple instrument is used for this purpose is called as spirometer, and the modified spirometer is called as the respirometer. Spirometer is made up of metal and the two chambers called as the outer and the inner chamber. Outer chamber is otherwise known as the water chamber because it filled with water and a floating drum is immersed within it. Inner chamber is inverted and has a small hole in the top and the long metal tube is passes through the inner tube from bottom to the top. Rubber tube is attached to the outer end of the metal tube. At the other end of the rubber tube the mouth piece is attached and by closing the nose with nose clip.

Spirometer is used for only the single breath. Repeated breathing is not indicated in this spirometer because repeated breathing can cause the occumulation of carbon dioxide and other air and oxygen are cannot be provided to the patients.

Computerized spirometer is the solid state does not contain drum or water column. Subjects are respire into a sophisticated transducer which is connected to the instrument of a cable.

Respirometer is the modified spirometer. It has the provision of removal of carbon dioxide and supply of the oxygen. Carbon dioxide is removed by placing soda lime inside the instrument from the oxygen cylinder by a suitable valve system. Oxygen is filled in the inverted drum above the water column and the subject can breath in and out with instrument for about 6minutes and recording can be done continuously[28, 29].

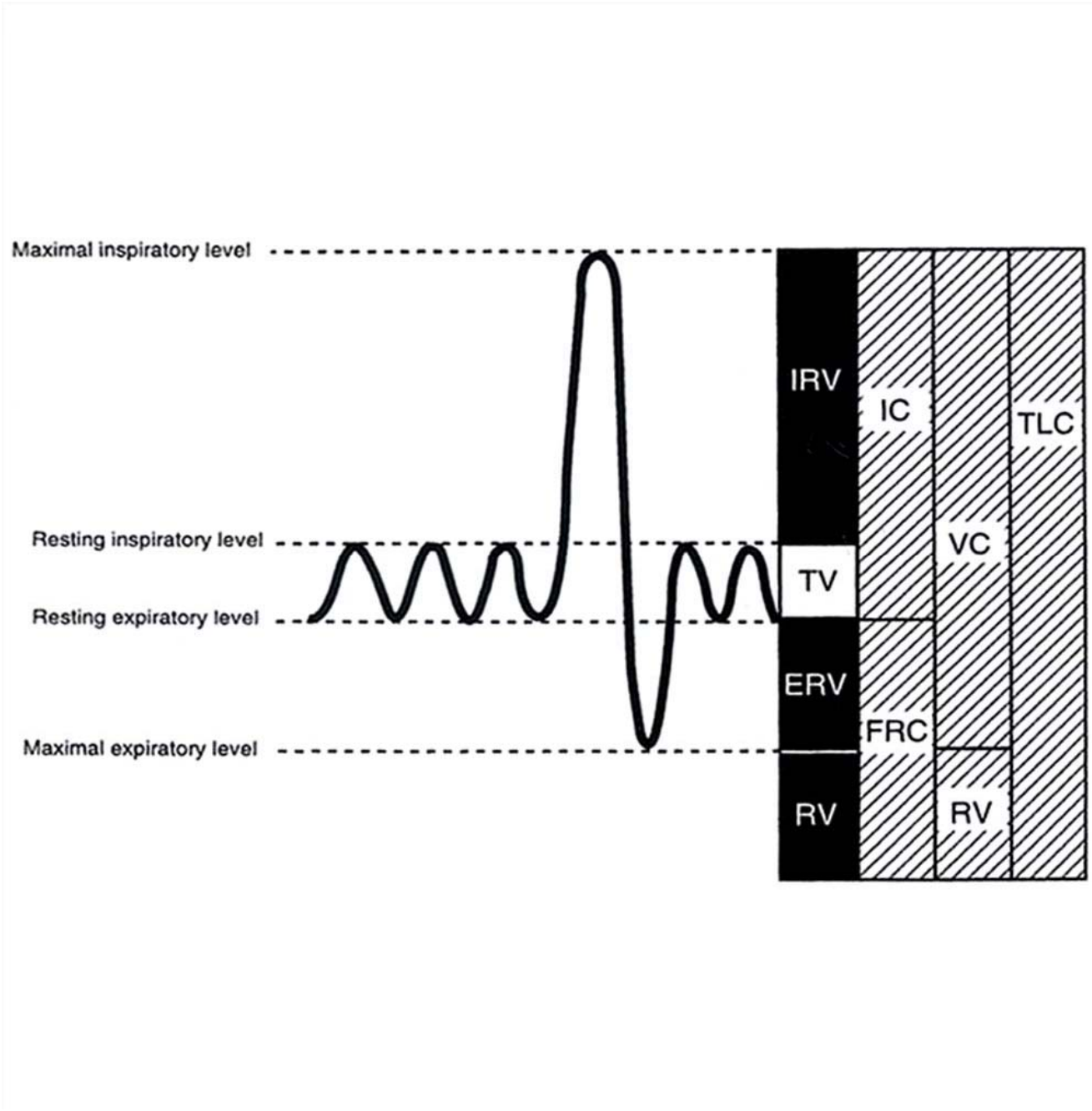
SPIROMETRY ACCEPTIBILITY CRITERIA:

1. There is a good starting of the test without any form of hesitation
2. No interrupting coughing and the closure of the glottis- may produces the faulty procedure
3. The flow will be constant, there should not be any variability in the flow of air
4. Not to terminate very earlier. It should be more than 6seconds
5. There should not be any leakage of air through the mouth or anywhere from the instrument
6. It should be reproducible in nature.

   The atleast two largest measurement of forced vital capacity and the two largest measurement of forced expiratory volume at the first second which was not exceeding more than 0.2 liter[30].

PFT:

LUNG VOLUMES AND CAPACITIES:



LUNG VOLUMES:

TIDAL VOLUME (TV):

It is the volume of air breathed in and out of the lungs in a single normal quiet breathing. It signifies the normal depth of breathing. Normal 500ml

Inspiratory Reserve Volume (IRV):

It is an additional volume of air that can be inspired forcefully after the end of normal inspiration.

3000ml

Expiratory Reserve Volume(ERV):

It is the additional amount of air that can be expired out forcefully after normal expiration. Normal 1500ml.

Residual Volume (RV):

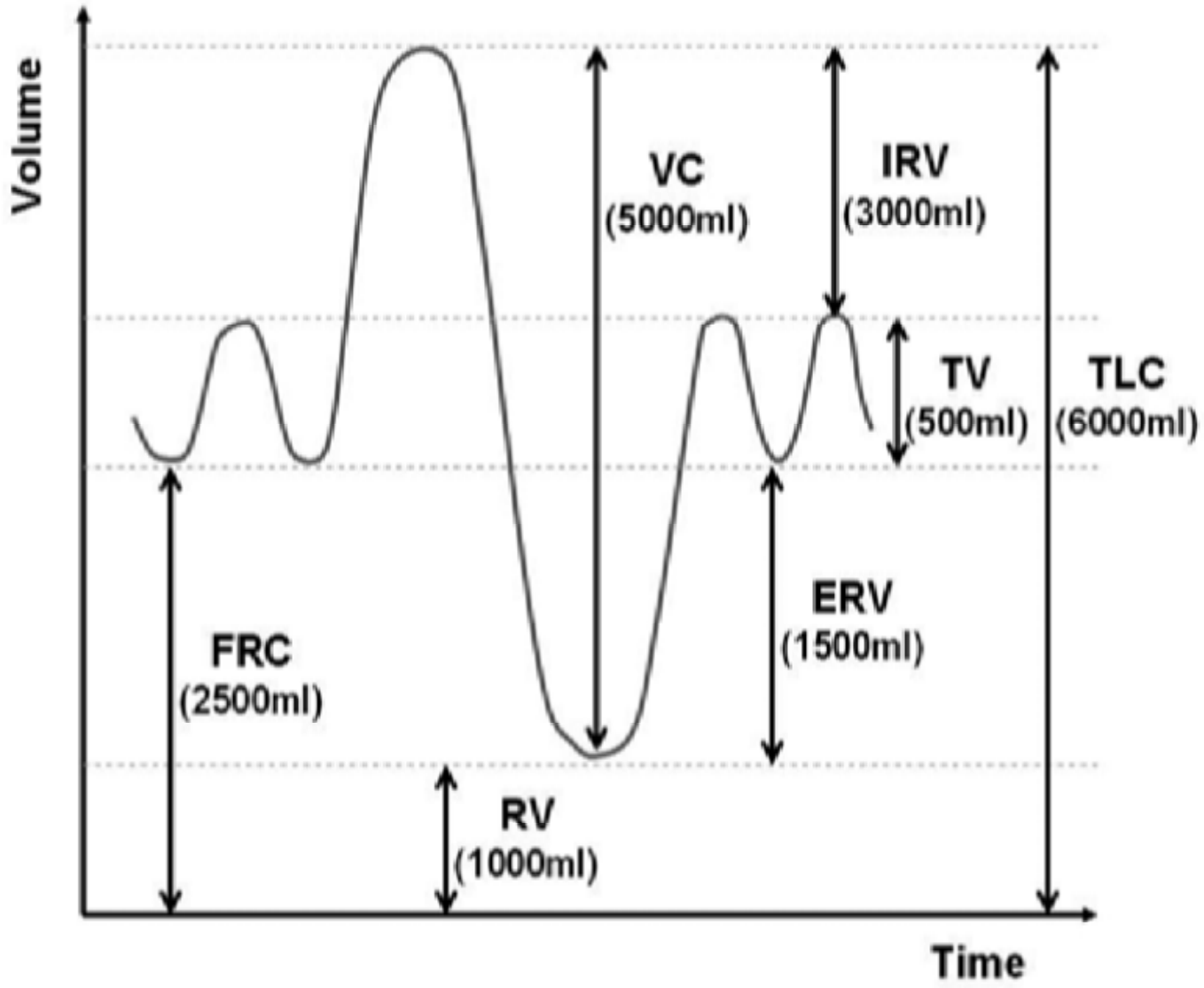
It is the volume of air remaining in the lungs even after forceful expiration.

Normal 1200ml

LUNG CAPACITIES:

Total Lung Capacity (TLC):

It is the volume of air present in lungs after a deep inspiration. It includes all the volumes (4-6 L).


Volume
VC
(5000ml)
IRV
(3000ml)
TV
(500ml)
TLC
(6000ml)
FRC
(2500ml)
ERV
(1500ml)
RV
(1000ml)
Time

Vital Capacity (VC):

It is the maximum volume of air that can be expelled out forcefully after a deep inspiration (60-70 ml/kg) 5000ml.

Inspiratory Capacity (IC):

It is the maximum volume of air that is inspired after normal expiration. (2400-3800ml).

Expiratory Capacity (EC):

TV+ ERV

Functional Residual Capacity (FRC):

It is the volume of air remaining in lungs after normal expiration (30-35 ml/kg) 2500 ml.

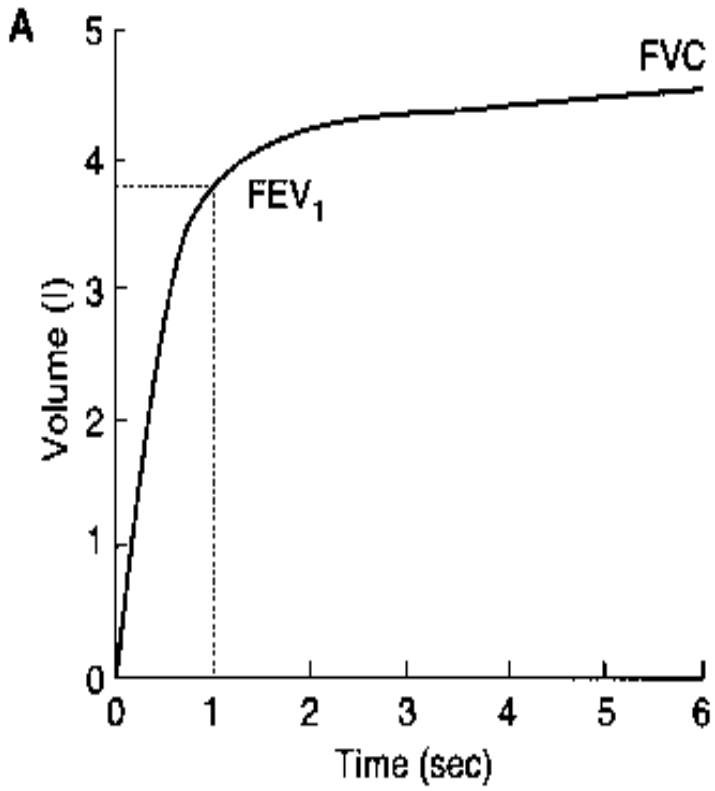
MEASURING RESIDUAL VOLUME AND FUNCTIONAL RESIDUAL CAPACITY:

1. Nitrogen washout technique.
2. Helium dilution method.
3. Body plethysmography

PROCEDURE:

With the patient comfortably seating with a closed circuit maneuver, first place the nose clip on and ask the patient to breath at the mouth piece and ask the patient to take a deep breath as much as fast and blow out as they hard enough till the technician asks the patient to stop.

FORCED VITAL CAPACITY (FVC):



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Total volume of air that can be exhaled forcefully from Total lung capacity.

The majority of FVC can be exhaled in <3 seconds in normal people, but often is much more prolonged in obstructive diseases measured in liters .

FORCED VITAL CAPACITY INTERPRETATION[30]:

Interpretation should be the percentage of the predicted value :

80-120percentage of predicted	Normal
70-79 percentage of predicted	Mild reduction
50%-69 percentage of predicted	Moderate reduction
<50 percentage of predicted	Severe reduction[30].

FORCED EXPIRATORY VOLUME IN 1st SECOND: (FEV_1)

Volume of air forcefully expired from full inflation (TLC) in the first second. Measured in liters (L) .Normal people can exhale more than 75-80% of their FVC in the first second; thus the FEV1/FVC can be utilized to characterize lung disease.

According to the Global initiative for lung disease criteria forced expiratory volume at the first second is the main diagnostic tool of diagnosing the obstructive lung disease[34] because the basic mechanism of these disorders are the end expiratory collapse of the alveoli. It is also used for therapy response

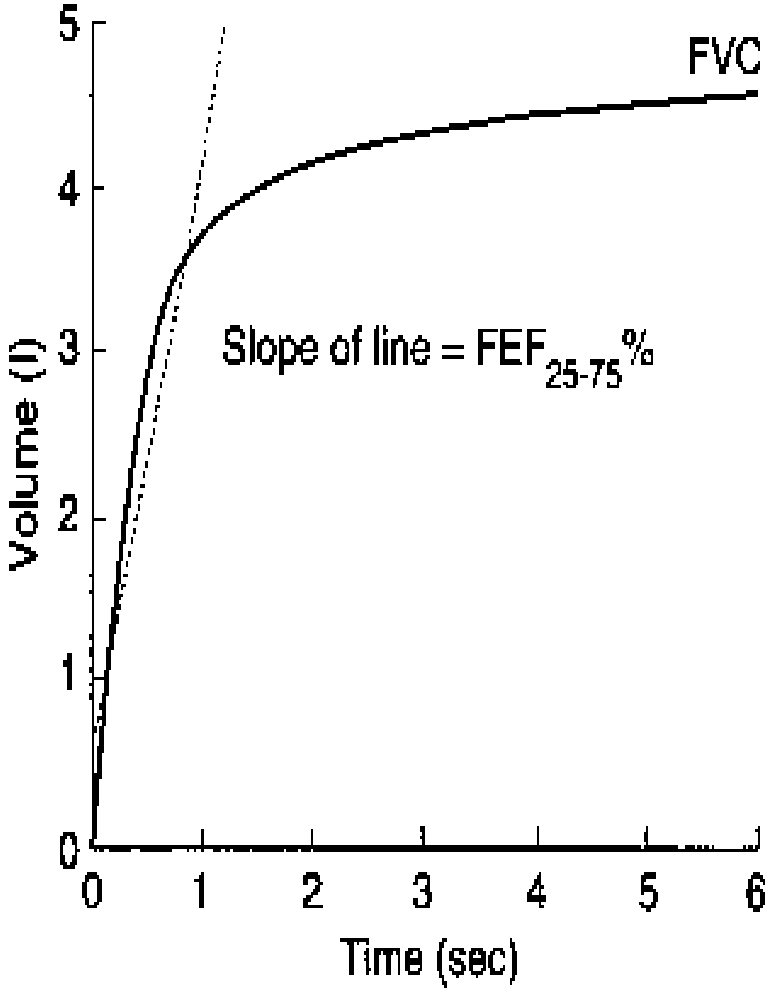
FORCED EXPIRATORY VOLUME IN 1st SECOND

INTERORETATION:

Interpretation of % predicted:

>75% of predicted	Normal
60%-75% of predicted	Mild obstruction
50-59% of predicted	Moderate obstruction
<49% of predicted	Severe obstruction[31]

FORCED EXPIRATORY FLOW 25-75% (FEF_{25-75})



Mean forced expiratory flow during middle half of Forced vital, capacity

Measured in Liter/second,

May reflect effort independent expiration and the status of the small airways,

Highly variable,

Depends heavily on forced vital capacity.

FORCED EXPIRATORY FLOW 25-75%(FEF_{25-75}) INTERPRETATION:

Interpretation of % predicted:

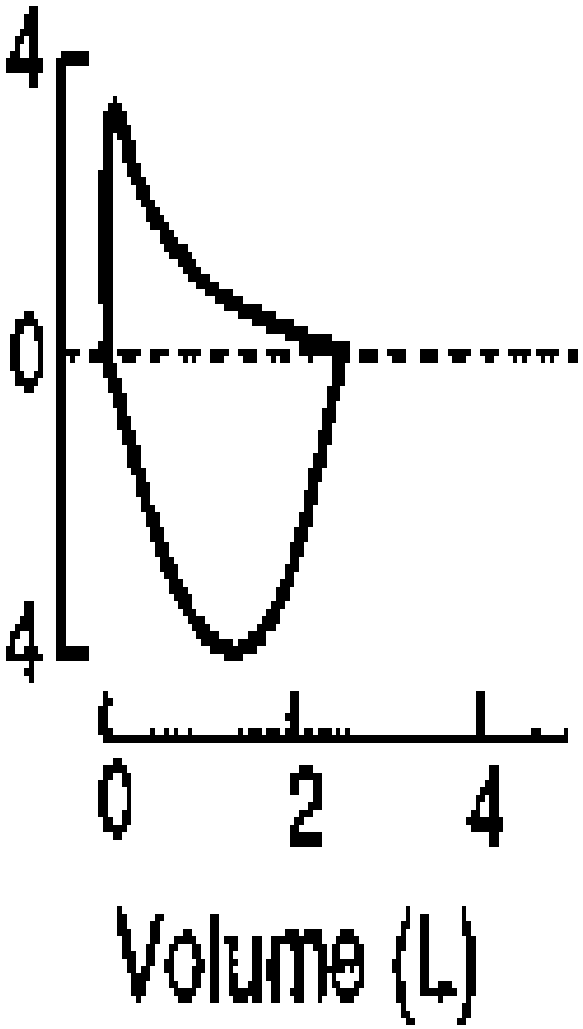
>60%of predicted	Normal
40-60% of predicted	Mild obstruction
20-40% of predicted	Moderate obstruction
<10% of predicted	Severe obstruction[31, 32]

OBSTRUCTIVE DISORDERS:

The main mechanism is limitation of expiratory airflow like asthma, COPD, bronchiolitis, bronchiectasis.

Manifested by decreased level of FEV_1, FEF_{25-75}, FEV_1/FVC ratio (<0.8)

Normal or increased total lung capacity.



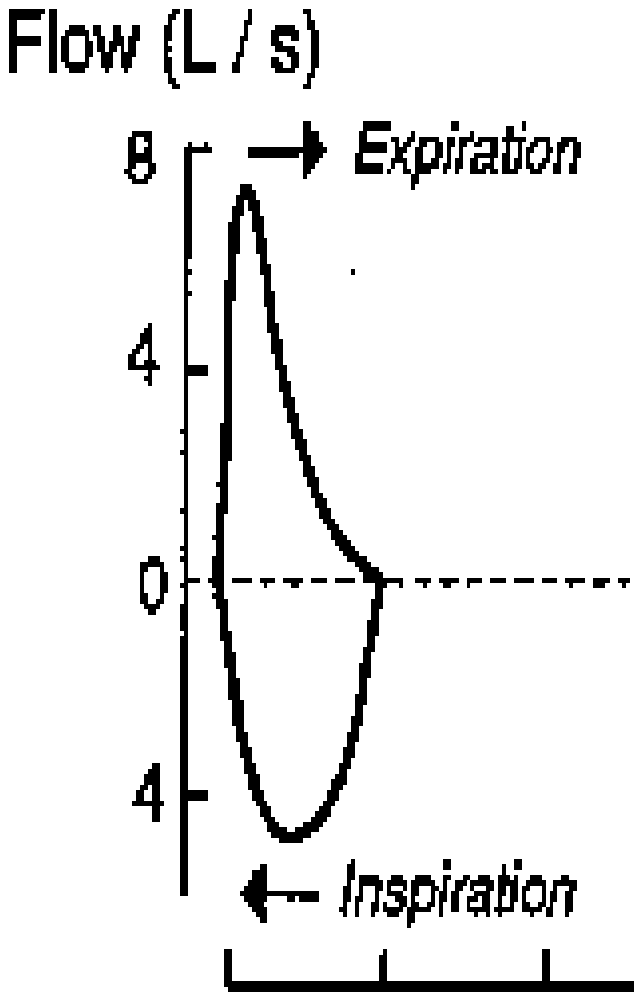
RESTRICTIVE LUNG DISEASE:

Manifested by decreased volumes of lungs because of:

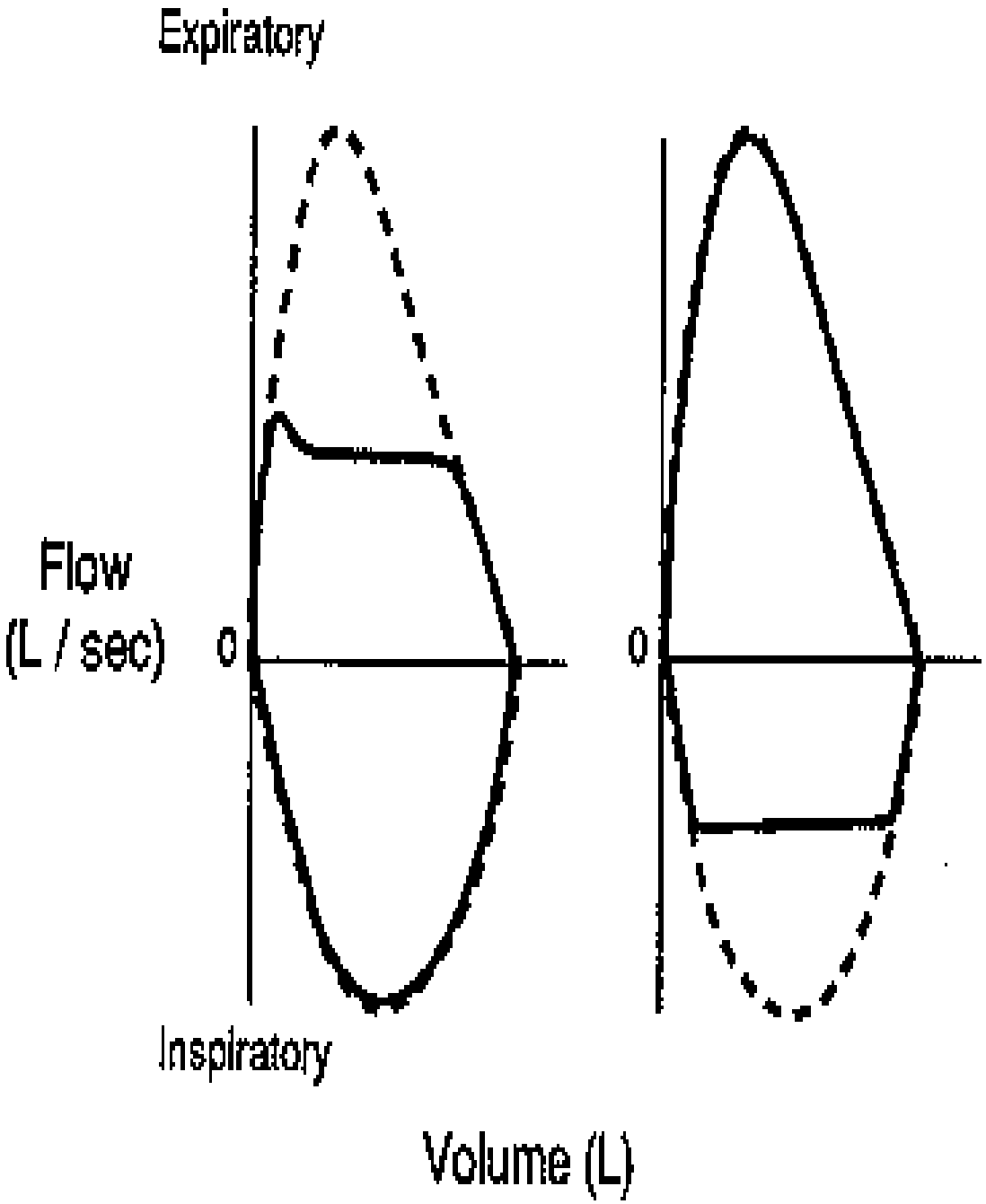
- abnormalities in lung parenchyma (interstitial lung disease)
- abnormalities in pleura, neuro muscular apparatus (e.g. muscular dystrophy) ,chest wall (e.g. scoliosis)

Decreased total lung capacity, forced vital capacity

Normal or increased FEV_1/FVC ratio

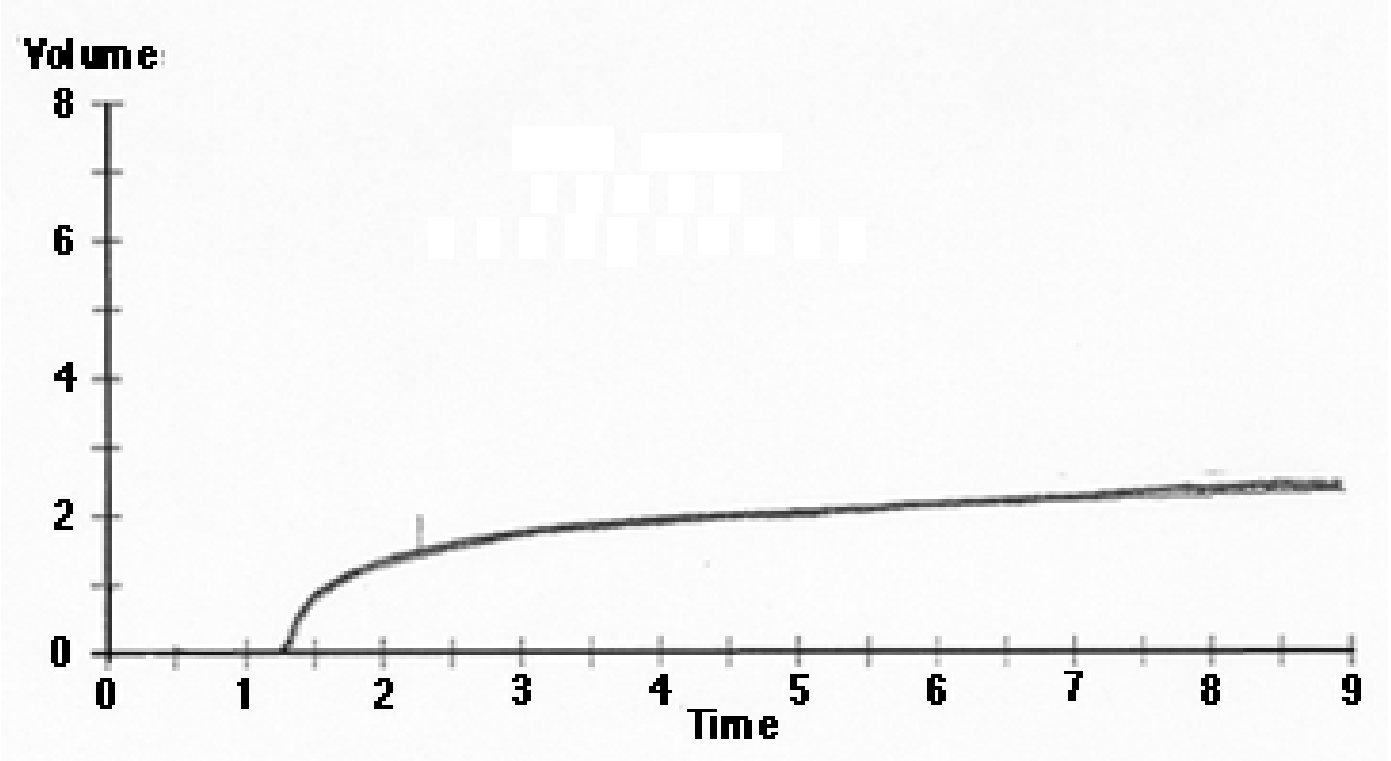


LARGE OBSTRUCTION IN AIRWAY:



Characterized by a truncated inspiratory or expiratory loop

OBSTRUCTIVE PATTERN:



FEV_1 is used to classifies the severity of COPD[33].

There is slow rise in the upstroke and it will not reaches the plateau line.

OBSTRUCTIVE LUNG DISEASE — DIFFERENTIAL DIAGNOSIS:

Asthma

COPD

chronic bronchitis

emphysema

Bronchiectasis

Bronchiolitis

Upper airway obstruction

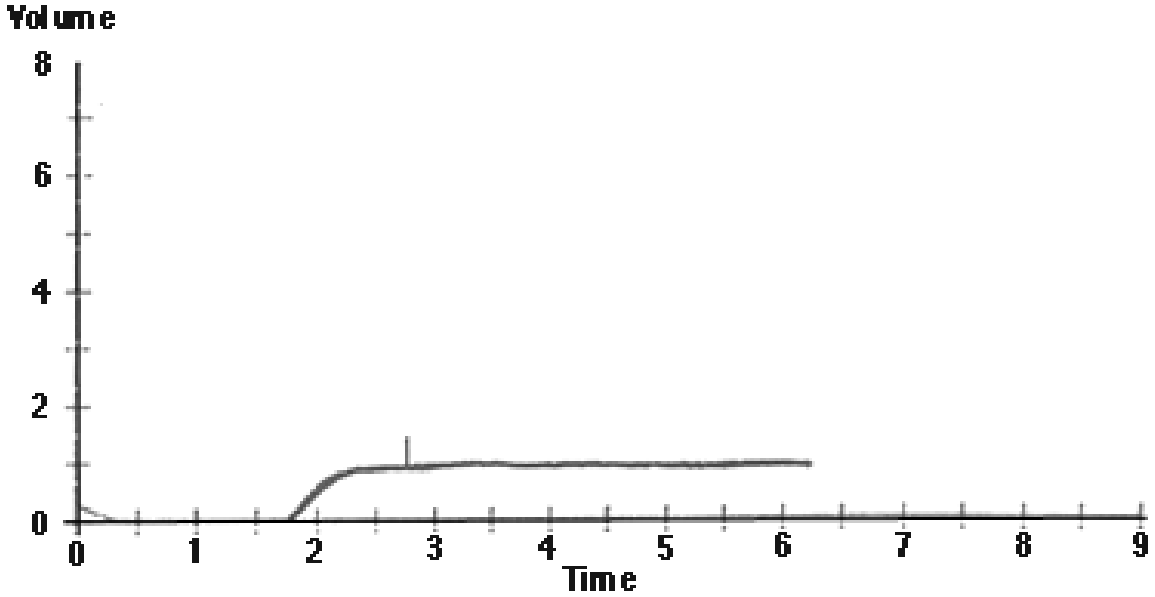
The obstructive lung disorders are mainly the disease of the abnormality in expiration[33]. Involves both the upper and lower respiratory tract.

RESTRICTIVE PATTERN:

Decreased FEV_1

Decreased FVC

FEV_1/FVC normal or increased



There is a rapid upstroke as in the normal volume[5, 8 ,23].

The plateau level is low

RESTRICTIVE LUNG DISEASE —DIFFERENTIAL DIAGNOSIS:

Chest wall

Neuromuscular

Pleural

Parenchymal

Restrictive lung diseases are mainly the abnormality in the inspiration and the expiration is not affected.

AIMS & OBJECTIVES

1.To compare the pulmonary function test in type 2 diabetics and non-diabetics a comparative study.

2. To evaluate the uncontrolled diabetes mellitus on lung functions and functional limitations of activities and pulmonary complications, in patients with type 2 diabetes.

MATERIALS AND METHODS:

STUDY POPULATION:

This study is to be conducted among 50 people with type 2 diabetes and 50 people with non-diabetes without having risk factors that affect the lung functions.

INCLUSION CRITERIA:

(i)People with type2 diabetes and non-diabetes in age group between 30 – 50 yrs

(ii)Both male and female

EXCLUSION CRITERIA :

(i) Patient refusal

(ii)Subjects with vertebral column or thoracic cavity anatomical abnormality

(iii) acute or chronic respiratory infections,

(iv) Neuro and muscular disease,

(v) Known cancer patients

(vi) Cardiac disease

(vii) Patient underwent major chest or abdominal surgeries

(viii) Smokers of any duration, betel nut chewers, smoking in any form of preparation using

(ix) Those who are obese

(x) Asthma and COPD

ANTICIPATED OUTCOME:

Type 2 diabetes with uncontrolled disease pattern will cause pulmonary complications mainly restrictive pattern of disease[1,5,6,14].

DATA COLLECTION:

The following information were collected from patients who were a known case of Diabetes mellitus and a recently noticed type 2 diabetes mellitus according to the American diabetes association criteria for diagnosing type 2 diabetes mellitus who attended medicine opd in the form of Age, Sex, Height, Weight, BMI, Random blood sugar, HBA1C value from both male and female with type 2 Diabetes mellitus those who were having symptoms of diabetes mellitus in a age between 30 to 50.

I have examined the patients properly and I have excluded those who were not fit for study according to my study exclusion criteria. All participants in my study underwent electrocardiography examination, echocardiogram examination, chest x ray examination, proper neurologic examination to eliminate those who were not fit for study.

I referred all the participants to department of thoracic medicine for pulmonary function testing. Before I refer the patients I have instructed the proper way of spirometric examination and procedure of the examination. I have collected the pulmonary function test results mainly FVC, FEV1, FEV1/FVC for all participants of 50 participants with type 2diabetes mellitus and 50 participants without type 2 diabetes mellitus.

LABORATORY INVESTIGATIONS:

- Random blood sugar
- HBA1C value
- FORCED VITAL CAPACITY
- FORCED EXPIRATORYVOLUME AT FIRST SECOND
- FEV1/FVC

DESIGN OF STUDY:

Cross sectional and Retrospective

PERIOD OF STUDY:

One year (September 2014 to september2015)

COLLBORATING DEPARTMENTS:

Department of Biochemistry

Department of Thoracic Medicine

ETHICAL CLEARANCE:

Obtained

CONSENT:

Individual written & informed consent

ANALYSIS:

Statistical Analysis

CONFLICT OF INTEREST:

Nil

FINANCIAL SUPPORT:

Nil

PARTICIPANTS:

50 persons with type 2 diabetes mellitus and 50 persons without type 2 diabetes mellitus.

STATISTICAL METHODS:

The data collected during the study was formulated into a master chart in Microsoft office excel and statistical analysis was done with help of computer using statistical software package SPSS V.17 for windows.

Using this software, frequencies, range, mean, standard deviation and ‘p’ were calculated through Student ‘t’ test, One way ANOVA, Pearson Correlation and Chi square test . P value of < 0.05 was taken as significant.

OBSERVATION AND RESULTS:

Table 1 : Age Profile

AGE	PATIENTS
<30	4%
31-35	25%
36-40	20%
41-45	30%
>46	21%

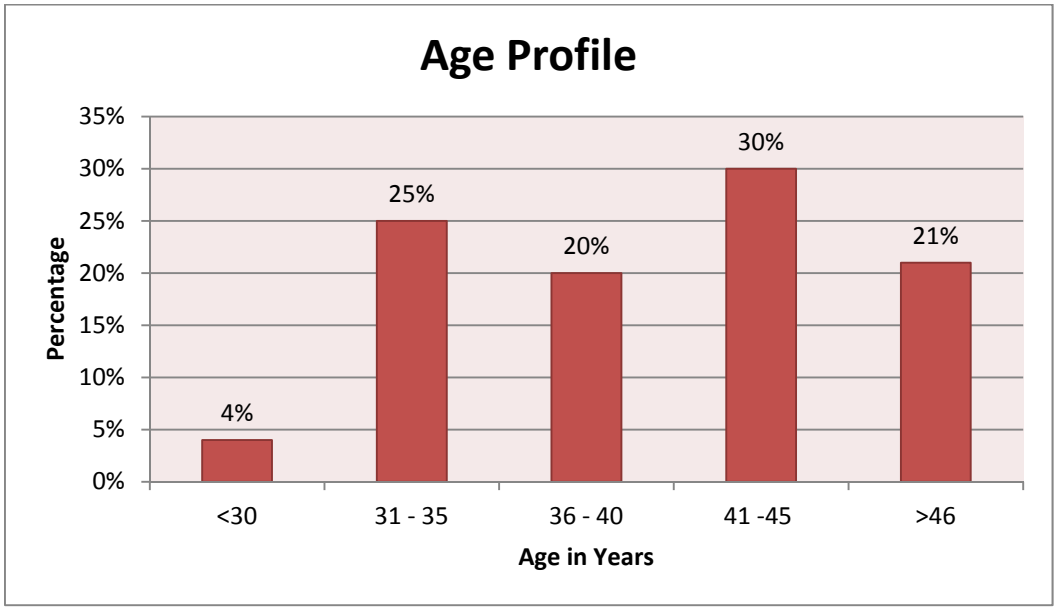


Fig.1 Age Profile

In our study majority of the population are in between the age group of 41 to 45.

GENDER DISTRIBUTION:

Table 2:

MALE	54%
FEMALE	46%

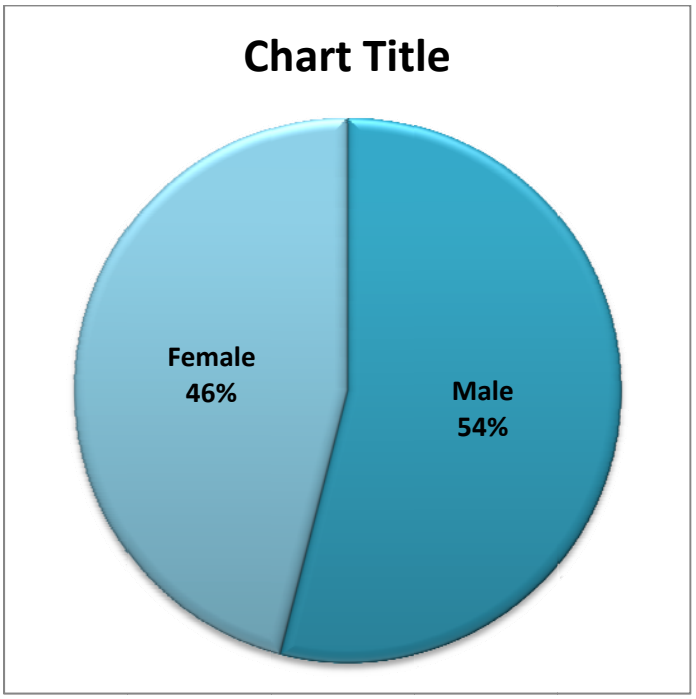


Figure 2 gender distribution

In our study the majority of patients are males.

Male participants around 54% and female participants around 46% in our study.

CASE DISTRIBUTION:

Table 3:

TYPE 2 DIABETICS	50
NON DIABETICS	50

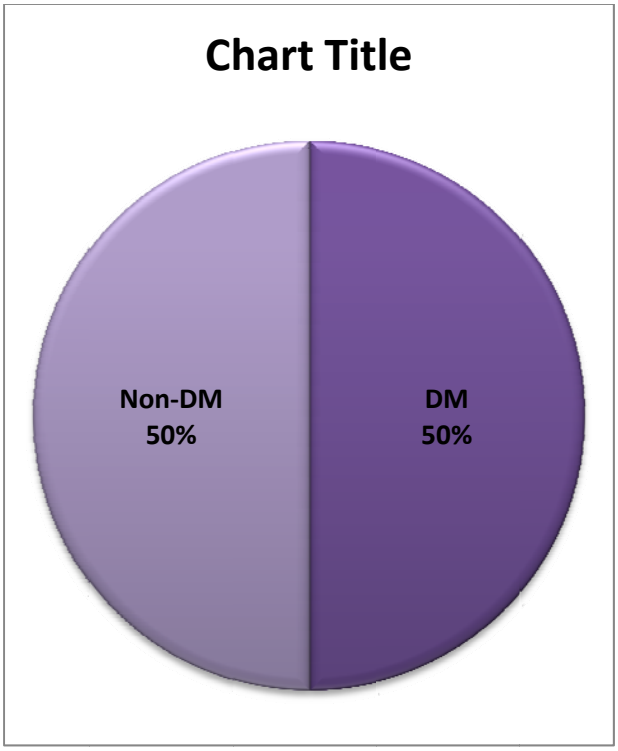


Figure 3 case distribution

In our study the type2 diabetic patients and non diabetics are equal in number.

Because we are comparing the study so it must be equal in number.

CORRELATION OF HBA1C AND PFT:

Table 4:

	PFT Coefficient correlation	P value
HBA1c	0.447	<0.0001

PEARSON CORRELATION TEST

In our study correlation between HBA1C and Pulmonary function is statistically significant (p value is <0.0001)

From this pearson correlation test we had came to the conclusion that there is a relationship between the HBA1C level and the results of the pulmonary function test. Most of the patients with the elevated levels had the abnormality in the pulmonary function tests mainly the restrictive pattern of the lung disease.From this there is surely a relation between the chronic uncontrolled diabetes mellitus and damage of the lungs.

COMPARISON OF PFT AMONG TWO GROUPS:

Table 5:

PARAMETERS	GROUP A (TYPE 2 DIABETICS) (n=50)		GROUP B (NON-DIABETICS) (n=50)		P VALUE
	MEAN	SD	MEAN	SD	
FEV1	68.37	11.19	77.27	13.47	0.001
FVC	68.18	13.25	84.52	6.33	<0.0001
FEV1 / FVC	100.66	13.86	90.8	15.46	0.001

INDEPENDENT SAMPLE t TEST

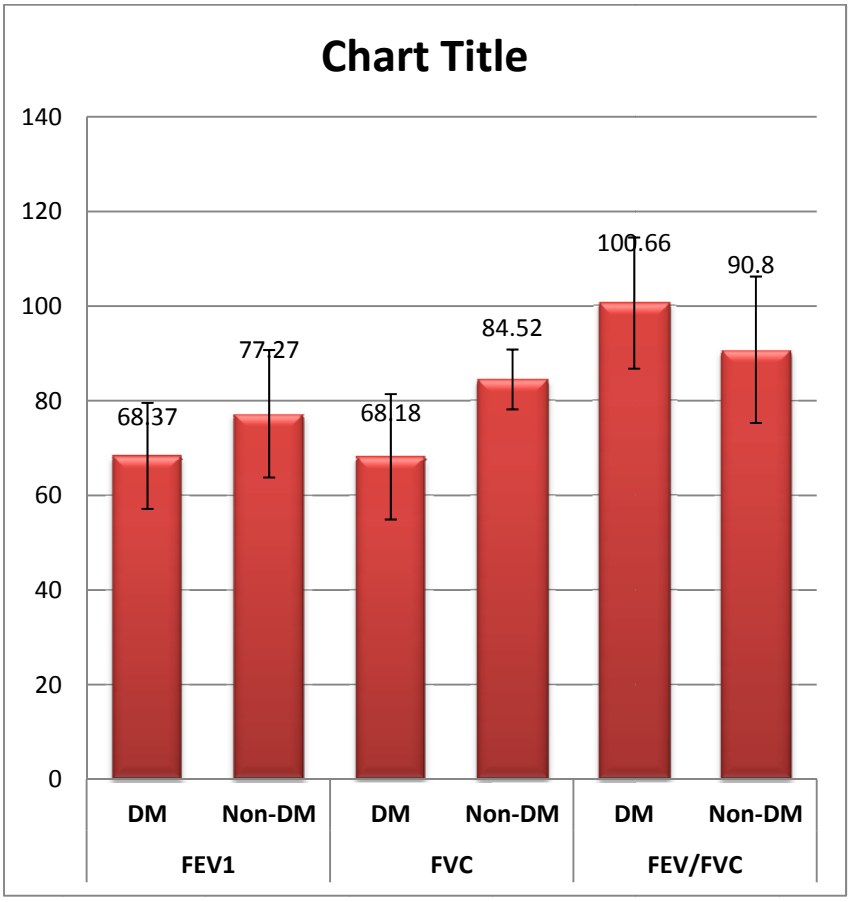


Figure 4 comparison of PFT

In our study both FEV1 and FVC are reduced, in which FVC have reduced more than FEV1 so that FEV1/FVC has increased, and the ‘p’value is significant in all the parameters according to independent sample t test.

This test depicts that most of the patients with type 2 diabetes mellitus having the restrictive pattern of the lung disease.

PATTERN OF PULMONARY FUNCTION:

Table 6:

	NORMAL	RESTRICTIVE PATTERN	OBSTRUCTION PATTERN
TYPE 2 DIABETICS	14%	80%	6%
NON-DIABETICS	78%	12%	10%

Pearson's chi square test

P<0.0001

In our study the patients with diabetes have 14% normal and 80% with restrictive lung disease and 6% with obstructive lung disease of pulmonary function tests.

In non-diabetics 78% having normal and 12% having restrictive pattern and 10 % having the obstructive pattern of lung disease.

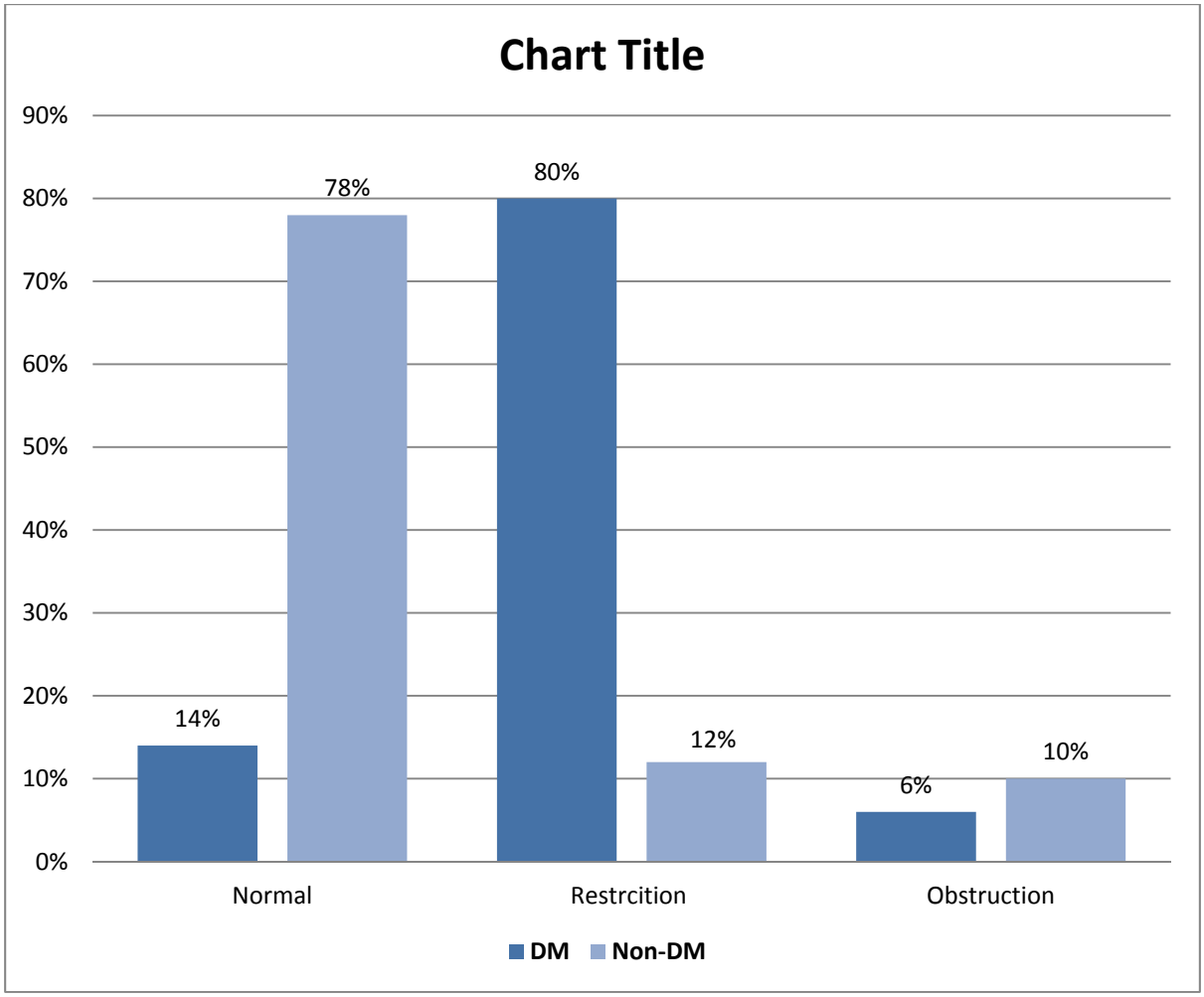


Figure 5 pattern of pulmonary function

According to our study profile the most of the people with type 2 diabetes mellitus having restrictive type of lung disease. About 80% having restrictive pattern and about 6% having obstructive pattern and about 14% having normal pattern. The relation between type 2 diabetes mellitus and restrictive pattern of lung disease is statistically significant.

DISCUSSION:

The present study has to assessed that type 2 diabetes was associated with reduced lung functions, by doing forced Spirometric Pulmonary Function Tests. This study clearly showed a highly statistically significant p value when the lung function tests (FVC, FEV1) were compared between type 2 diabetics and controls (age, sex and BMI matched).

In our study done in Kanyakumari medical college hospitals, about 100 participants were studied of which50 are having type 2 diabetes mellitus are cases and 50 without diabetes mellitus are controls and the males are 54 and females are 46 in number. The age distribution in our study mostly between 41to45 are 30%. Pulmonary function test has been conducted in all the persons.

There is statistically significant value in correlation between HBA1c and pulmonary function test results were noted and the p value is <0.0001.The FEV1and FVC both are reduced in type2 diabetic patients and the FEV1/FVC is increased. Mean, standard deviation are decreased in type 2 diabetics when comparing to non-diabetics. The p value foe FEV1 in type 2 diabetics is0.001 and foe FVC is <0.0001 and for FEV1/FVC is 0.001.

The pattern of lung disease is mostly restrictive pattern in patients having type 2 diabetes mellitus. Only 3 person's with diabetes having obstructive type of disease. And the p value is <0.0001 is statistically significant.

Recent studies which were conducted by Lange et al., indicated that the type 2 diabetic patients are having mild decrease in forced vital capacity may because of impaired immunity against environmental challenges such as infections in diabetes and smoking. In a study which was done by Davis[1,2,3,5]. A Wendy et al., it was found that there was a decrease in mean FVC values in type 2 diabetics. In a study which was done by Robert E. Walter et. al., it was found that there was a progressive decrease in mean forced vital capacity value is 109 ml/year [33,34].

The FEV1/FVC % was increased in type 2 diabetics as compared to that in the controls and the increase was statistically significant. The increased FEV1/FVC % suggested that the impairment of pulmonary functions in type 2 diabetics was primarily restrictive in nature

Davis et al., study detected that almost all the parameters like forced vital capacity, forced expiratory volume at 1st second are decreased so that the ratio is increased almost most of the patient with uncontrolled diabetes mellitus[23,27]. So it is clear that type 2 diabetes mellitus affect the lung and lung may the target organ for damage and the pattern of disease is restrictive in nature[1,4,5,9].

Although the underlying mechanisms which relate type 2diabetes to reduced lung functions remains unclear, previous studies have suggested several possible explanations, which include glycosylation of chest wall and bronchial tree proteins and increased cross-linkage formation between polypeptides of collagen in pulmonary connective tissue, which decrease Forced vital

capacity,basal lamina thickening, and increased susceptibility to respiratory infections[1,3,9,35].

The study which was done by Mario Cazzola et al., on human isolated bronchi elucidated the obstructive nature of pulmonary pathology in diabetes at a molecular level[16, 18]. Thus hyperglycaemia may contribute to obstruction of airways.

The lungs are affected by diabetic microangiopathy[33, 34]. This was evidenced autopsy findings in human diabetic subjects, which showed pulmonary microangiopathy, thickening of alveolar epithelia, pulmonary capillary basal lamina thickening, centrilobular emphysema, and , thickening of alveolar epithelia. Type 2 Diabetes mellitus can cause the development of pulmonary complications due to collagen and elastin changes as well as microangiopathy[1, 2, 34, 35].

LIMITATION:

1. Few studies showed there is no correlation between HBA1C and PFT's.

2. Sample size is small

3. Patients refusal

4. There is no explained exact mechanisms of restrictive lung pattern of lung disease in type 2 diabetics.

CONCLUSION:

The findings from our study is nearly related with other studies that have done in the diabetics pulmonary function test. It is clearly shown that diabetes will affect the lung too that too mainly restrictive pattern of lung disease is formed but some study shows obstructive pattern also.

Now it is clear that lung may the target organ in diabetes mellitus. Chronic uncontrolled diabetes is the leading cause of lung complications. So an intensive management will decrease the rate of death by an improved ventilatory function. pulmonary dysfunction may one of the earliest and easiest measurable non– metabolic variation in diabetes, so the diabetic patients should undergo pulmonary function testing along with other tests.

So it has been assigned that patients with type 2 diabetes mellitus should undergo pulmonary function tests intermittently .This will help to assess the pulmonary complication in type 2 diabetes mellitus and to detect in the earlier stages itself. But it needs studies to assess the clean mechanism behind abnormality in pulmonary function test in the patients with type 2 diabetes mellitus .

SUMMARY:

The pulmonary complications of type 2 diabetes mellitus are rare only. Our study assessed that the comparison of pulmonary function test in type 2 diabetics and non-diabetics. This study has to assess the effects of uncontrolled diabetes mellitus on lung functions.

100 participants were selected on which 50 having type 2 diabetes mellitus and 50 without diabetes mellitus in a age group between 30to50. Sex, height , weight, BMI,HBA1C,FEV1,FVC,FEV1/FVC were analysed in all the participants. 54 are males and 46 are females.

Cases with elevated HBA1c will affect the lung function. The FEV1 and FVC are reduced so that FEV1/FVC is increased in our study shown that producing restrictive type of lung disease. About 80% of people in type 2 diabetics having restrictive pattern of lung disease and only 6% having obstructive type lung disease.

So it is clearly shown that chronic hyperglycemia is affect the lung functions mainly mechanical lung functions. And the nature of lung disease is restrictive type, so the patient with type 2 diabetes mellitus will check the pulmonary function tests intermittently along with other tests to reduce the morbidity and mortality from the pulmonary complications.

ANNEXURE

BIBLIOGRAPHY

1. Viberti GC, Rosiglitazone. Potential beneficial impact on cardiovascular disease. Int. J. ClincPract. 2003; 57 (2): 128-34
2. Boulbou MS, Gourgoulianis KI, Klisiaris VK, Tsikrikas TS, Stathakis NE, Molyvdas PA. Diabetes mellitus and lung function. Med Princ.Pract. 2003;12(2): 87-91.
3. Recommendations for a standard technique—1995 update. Am J RespirCrit Care Med.1995;152:2185-98
4. Asanuma Y, Fujiya S, Ide H, Agishi Y. Characteristics of pulmonary function in patients with diabetes mellitus. Diabetes. Res. Clin. Pract. 1985; 1(2): 95-101.
5. Lange P, Groth S, Kastrup J, Mortensen J, Appleyard M, Nyboe J, et al. Diabetes mellitus, plasma glucose and lung function in a cross-sectional population study. Eur. Respir J. 1989;2 (1):14-19.
6. Benbassat CA, Stern E, Kramer M, Lebzelter J, Blum I, Fink G. Pulmonary function in patients with diabetes mellitus. Am. J. Med. Sci. 2001;322 (3): 127-32.
7. Sreeja CK, Elizabeth Samuel, C Kesavachandran, Shankar Shashidhar. Pulmonary function in patients with Diabetes Mellitus.Indian J PhysiolPharmacol. 2003;47 (1):87 – 93.

8. P Lange, J Parner, P Schnohr, G Jensen. Copenhagen City Heart Study: longitudinal analysis of ventilatory capacity in diabetic and non-diabetic adults. EurRspir J. 2002;20:1406 -12.
9. Davis WA, Knuiman M, Kendall P, Grange V, Davis TM. Fremantle Diabetes Study.Diabetes Care. 2004;27(3):752-7. 22.
10. Walter R, Beiser A, Rachel J et.al., Association between glycemic state and lung function. The Framingham Heart Study. Am J RespirCrit Care Med. 2003 ; 167 : 911-16.
11. Davis Timothy ME, Mathew Knuimann, Peter Kendall. Reduced pulmonary function and its association in type-2 Diabetes.Diabetes Res ClinPract.2000; 50: 152-59.
12. Sandler M. Is the lung a 'target organ' in diabetes mellitus? Arch Intern Med. 1990; 150(7):1385-88
13. ShravyaKeerthi G, Sharan B Singh M, Hari Krishna Bandi, Suresh M, Preetham J K, Mallikarjuna Reddy. Deterioration of Pulmonary Functions in Type 2 Diabetes Mellitus.IOSR.Journal of Pharmacy and Biological Sciences (IOSRJPBS). 2012; 1 (1) : 39-43.
14. Weynand B, Jonckheere A, Frans A, Rahier J. Diabetes mellitus induces a thickening of the pulmonary basal lamina. Respiration.1999; 66:14–1
15. Fine MJ, Smith MA, Carson CA, Mutha SS, Sankey SS, Weissfeld LA, et al. Prognosis and outcomes of patients with community-acquired pneumonia: a meta-analysis. JAMA. 1996;275:134–41.

16. Mario Cazzola, LuiginoCalzetta, Paola Rogliani, DavideLauro, Lucia Novelli, Clive P, Varsha et al. High Glucose Enhances Responsiveness of Human Airways Smooth Muscle via the Rho/ROCK Pathway. Am J Respir Cell Mol Biol. 2012; 47 (4): 509-16.

17. Barnes PJ. The role of inflammation and anti-inflammatory medication in asthma.Respir Med 96 (Suppl. A). 2002;13:211–15.

18. Rodriguez M, Guerrero R. Increased levels of C-reactive protein in non-controlled type 2 diabetic subjects. J Diabetes Complications.1999; 13:211–15.

19. KaminskyDA.Spirometry and Diabetes.Diabetes Care. 2004; 27: 837-3

20. World Health Organization. Fact sheet: Diabetes. No. 312, November 2008. Available from: htpp://www.who.int/mediacentre/factsheets/fs312/ en/index.html. [Last accessed on 2009 Apr 14].

21. King H, Aubert RE, Herman WH. Global burden of diabetes 1995 to 2025. Prevalence, numerical estimates and projections. Diabetes Care 1998;21:1414–31.

22. Sandler M. Is the lung is target organ in diabetes mellitus? Arch Intern Med 1990;150:1385–8.

23. Klein OL, Krishnan JA, Jlick S, Smith LJ. Systematic review of association between lung function and type 2 diabetes mellitus.

24. Hamlin CR, Kohn RR, Luschin JH. Apparent accelerated aging of human

collagen in diabetes mellitus. Diabetes 1975;24:902–4.

25. Fogarty AW, Jones S, Britton JR, Lewis SA, McKeever TM. Systemic inflammation and decline in lung function in a general population: A prospective study. Thorax 2007;62:515–20.

26. Mori H, Okubo M, Okamura M, Yamane K, Kado S, Egusa G, *et al*. Abnormalities of pulmonary function in patients with non insulin dependent diabetes mellitus. Intern Med 1992;31:189–93.

27. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, et al. Standardization of spirometry. Eur Respir J 2005;26:319–38.

28. Nathan DM, Singer DE, Hurxthal K, Goodson JD. The clinical information value of the glycosylated haemoglobin assay. N Engl J Med 1984;310:341–6.

29. Davis WA, Knuiman M, Kendall P, Grange V, Davis TM. Glycemic exposure is associated with reduced pulmonary function in type 2 diabetes, the fremantle diabetes study. Diabetes Care 2004;27:752–7.

30.Asanuma Y, Fujiya S, Ide H, Agishi Y. Characteristics of pulmonary function in patients with diabetes mellitus. Diabetes Res Clin Pract 1985;1:95–101.

31.Lange P, Groth S, Kastrup J, Mortensen J, Appleyard M, Nyboe J, et al.

32. Barrett–Conor E, Frette C. NIDDM, impaired glucose tolerance, and pulmonary function in older adults. Diabetes Care 1996;19:1441–4.

33. Davis TM, Knuiman M, Kendall P, Vu H, Davis WA. Reduced

pulmonary function and its association in type 2 diabetes: The fremantle diabetes study. Diabetes Res Clin Pract 2000;50:153–9.

34. Engstrom GJ, Janzon L. Risk of developing diabetes is inversely Related to lung function: A population based cohort study. Diabet Med 2002;19:167–70.

35. Yeh HC, Punjabi NM, Wang NY, Pankow J, Duncan BB, Cox CE,et al

PROFORMA:

Name : Age/Sex: Occupation:

Presenting complaints:

Past history:

H/o DM, HIV, PT, HT, CKD, CVD, COPD etc

Clinical examination:

General examination:

Consciousness, Pallor, jaundice, Clubbing, Lymphadenopathy, hydration status

Vitals: PR, BP,Temp, RR, SpO2

Systemic examination:

CVS:

RS:

Abdomen:

CNS:

Laboratory investigations:

Random blood sugar

Pulmonary function test

MASTER CHART:

GROUP – I (TYPE 2 DIABETICS)

S No	Name	Age (yr)	Sex	RBS (mg/dl)	HbA 1c	FEV1 (% of predicted)	FVC (% of predicted)	FEV1 / FVC	Result
1	RAMASAMY	50	M	350	8.2	60	54	90	Mod restriction
2	ROSAMMAL	40	F	208	7.9	70	65	100	Mild restriction
3	RANI	32	F	285	7.5	87	89	97	Normal
4	BHAKIYAM	48	F	265	6.9	67	63	106	Mod restriction
5	SIVAPRAKASAM	50	M	325	9.4	75	70	107	Mild restriction
6	SAROJINI	41	F	300	8.5	80	75	106	Mild restriction
7	AMEERA	36	F	313	8.7	77	72	106	Mild restriction
8	CLITUS	45	M	420	8.2	55	50	110	Mod restriction
9	GEORGE	33	M	365	7.8	81	88	92	Normal
10	RAVI	40	M	215	6.9	78	74	105	Mild restriction
11	SUJIN	49	M	235	7.2	57	55	103	Mod restriction
12	STEPHEN	32	M	256	6.7	85	88	96	Normal
13	DANIEL	44	M	260	7.9	65	65	100	Mod restriction
14	VELAMMAL	45	F	280	8.6	55	81	67	Mild obstruction
15	REEMA	47	F	274	9.2	80	76	105	Mild restriction

S No	Name	Age (yr)	Sex	RBS (mg/dl)	HbA 1c	FEV1 (% of predicted)	FVC (% of predicted)	FEV1 / FVC	Result
16	JESINTHA	38	F	245	10.0	66.90	66	101	Mild restriction
17	KALIAMMAL	35	F	210	9.9	50	48	104	Severe restriction
18	MURUGAPPAN	30	M	234	7.6	60	45	133	Severe restriction
19	PALANI	43	M	212	7.4	60	56	107	Mod restriction
20	MUTHURATHINAM	41	M	256	7.9	75	72	104	Mild restriction
21	RAJAN	33	M	190	8.9	50	41	121	Severe restriction
22	VEERAIYAN	36	M	265	8.4	66	64	103	Mod restriction
23	KARUPPAIAH	50	M	211	8.6	62	90	68	Mild restriction
24	JANAKI	36	F	305	9.2	59	55	107	Mod restriction
25	CHRISTHUDHAS	43	M	194	9.9	50	48	104	Severe restriction
26	CHANDRU	32	M	222	10.2	73	69	105	Mod restriction
27	JOSEPH	30	M	245	7.6	82	88	93	Normal
28	HELEN	50	F	198	11.2	65	47	138	Severe restriction
29	ANTONY	47	M	200	8.3	50	52	96	Mod restriction
30	LILLY BHAI	49	F	267	8.8	62	55	112	Mod restriction

S No	Name	Age (yr)	Sex	RBS (mg/dl)	HbA 1c	FEV1 (% of predicted)	FVC (% of predicted)	FEV1 / FVC	Result
31	SARASWATHI	37	F	287	11.3	88	90	97	Normal
32	SHEELA	39	F	234	7.9	86	88	97	Normal
33	FATHIMA	34	F	265	8.5	75	80	97	Mild restriction
34	LAKSHMI	44	F	218	8.9	65	67	97	Mod restriction
35	SELVANAYAKI	43	F	143	8.6	70	66	106	Mod restriction
36	RADHAMANI	50	F	167	9.9	50	78	64	Mod restriction
37	KANAGAM	43	F	219	9.5	70	74	94	Mild restriction
38	VISALAM	32	F	112	8.3	75	70	107	Mild restriction
39	VIGNESH	39	M	178	8.4	70	74	94	Mild restriction
40	JOEL	37	M	233	9.5	87	89	97	Normal
41	SATHISH	42	M	245	9.7	75	71	105	Mild restriction
42	ELANKUMARAN	32	M	287	9.2	55	59	93	Mod restriction
43	GANESH	41	M	166	10.4	70	60	116	Mod restriction
44	SURESH	50	M	240	10.7	70	56	125	Mod restriction
45	DHANALAKSHMI	34	F	259	7.5	65	70	92	Mild restriction

S No	Name	Age (yr)	Sex	RBS (mg/dl)	HbA 1c	FEV1 (% of predicted)	FVC (% of predicted)	FEV1 / FVC	Result
46	SUBHA	30	F	157	6.7	81	80	87	Mild restriction
47	THANGAMMAL	41	F	188	6.2	80	74	108	Mild restriction
48	NAZRIN	44	F	299	6.7	69	66	104	Mild restriction
49	ABBAS	47	M	350	8.8	50	66	75	Mild restriction
50	RAJESHWARI	46	F	300	6.8	65	70	92	Mild restriction

GROUP – II (NON-DIABETICS)

S No	Name	Age (yr)	Sex	RBS (mg/dl)	HbA 1c	FEV1 (% of predicted)	FVC (% of predicted)	FEV1 / FVC	Result
1	KOSALAI	31	F	111	5.5	83	84	98	Normal
2	SRIKUMARI	33	F	120	5.1	70	74	94	Mild restriction
3	MANIBHARATHI	33	M	99	4.9	81	80	87	Mild restriction
4	PAAPA	36	F	80	4.6	84	90	99	Normal
5	VASANTHA	39	F	97	4.1	81	88	92	Normal
6	SHEIKH MEERAN	46	M	94	5.2	82	85	96	Normal
7	THAVASI	38	M	112	5.5	83	88	94	Normal
8	SUDHAKAR	43	M	130	5.6	50	66	75	Mild restriction
9	ARUNACHALAM	33	M	134	4.9	86	89	96	Normal
10	SINGARI	30	F	95	4.3	91	92	98	Normal
11	AMIRDHAKALA	43	F	140	4.6	90	92	97	Normal
12	MUTHURATHINAM	44	M	123	5.1	55	90	61	Mod obstruction
13	CHIDAMBARAM	45	M	113	5.3	80	81	98	Normal
14	VIJAYAN	50	M	116	4.7	83	85	97	Normal
15	ESSAKIPILLAI	41	M	98	4.2	80	82	97	Normal
16	CHINNAPOO	34	M	87	4.6	45	84	53	Mod obstruction

17	KUNJAMMAL	38	F	99	5.5	83	85	97	Normal
18	FILOMENA	40	F	100	5.2	88	89	98	Normal
19	SUBBAIYAH	41	M	101	5.3	82	84	97	Normal
20	GANDHI	32	M	96	4.2	787	86	90	Normal
21	PICHUMANI	31	M	88	4.5	87	87	100	Normal
22	ANNAPANDI	33	M	83	4.4	44	89	49	Mod obstruction
23	KOLAPPAN	45	M	101	4.3	82	85	96	Normal
24	VIJAYARANI	50	F	110	5.1	84	89	94	Normal
25	JENNIFER	43	F	111	5.4	80	82	97	Normal
26	KANNAN	32	M	130	5.3	81	84	96	Normal
27	SAHAYAM	31	M	126	4.5	82	86	95	Normal
28	RATHINAKUMAR	45	M	117	4.2	84	88	95	Normal
29	IYAPPAN	46	M	128	4.8	85	89	96	Normal
30	BHAIRAVI	36	F	98	4.9	83	77	107	Mild restriction

S No	Name	Age (yr)	Sex	RBS (mg/dl)	HbA 1c	FEV1 (% of predicted)	FVC (% of predicted)	FEV1 / FVC	Result
31	BELLA	38	F	91	5.6	81	87	93	Normal
32	KAMALAIYAN	41	M	82	5.2	84	88	95	Normal
33	NAGAPPAN	31	M	79	5.3	43	85	50	Mod obstruction
34	LOURDHUMARI	33	F	115	4.3	76	83	91	Normal
35	VEERAPRABU	43	M	112	4.6	80	81	98	Normal
36	JAYAN	31	M	78	4.2	85	89	96	Normal
37	THIRUMALAI	45	M	99	4.5	58	88	65	Mild obstruction
38	CHELLAIYAN	44	M	116	5.4	89	89	100	Normal
39	KANTHARI	40	F	130	5.4	84	86	97	Normal
40	FRANCIS	37	M	132	5.1	85	85	100	Normal
41	JENILA	36	F	125	4.2	79	88	89	Normal
42	SELVAM	34	M	115	4.7	65	54	120	Mod restriction
43	CHELLATHAI	50	F	112	4.9	80	82	97	Normal
44	RAJI	49	F	134	5.5	87	89	97	Normal
45	MICHAEL	43	M	101	5.4	80	84	95	Normal

S No	Name	Age (yr)	Sex	RBS (mg/dl)	HbA 1c	FEV1 (% of predicted)	FVC (% of predicted)	FEV1 / FVC	Result
46	RAJAN	48	M	91	4.9	84	86	97	Normal
47	BOOMADEVI	43	F	96	4.1	82	87	94	Normal
48	THANGAKANI	31	F	78	4.7	32	82	39	Severe restriction
49	KANNAGI	50	F	124	5.0	79	81	97	Normal
50	ARULTHANGAM	45	F	129	5.2	80	82	97	Normal

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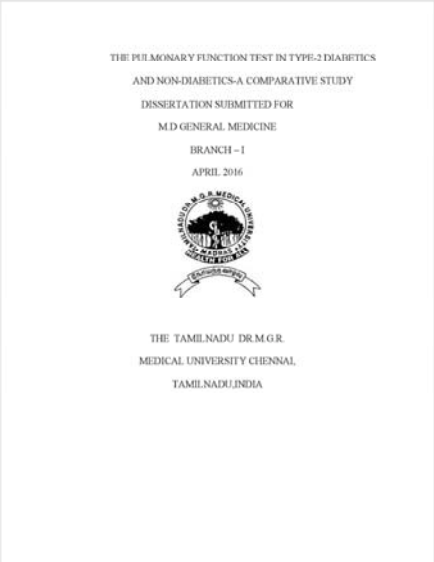

THE PULMONARY FUNCTION TEST IN TYPE-2 DIABETICS
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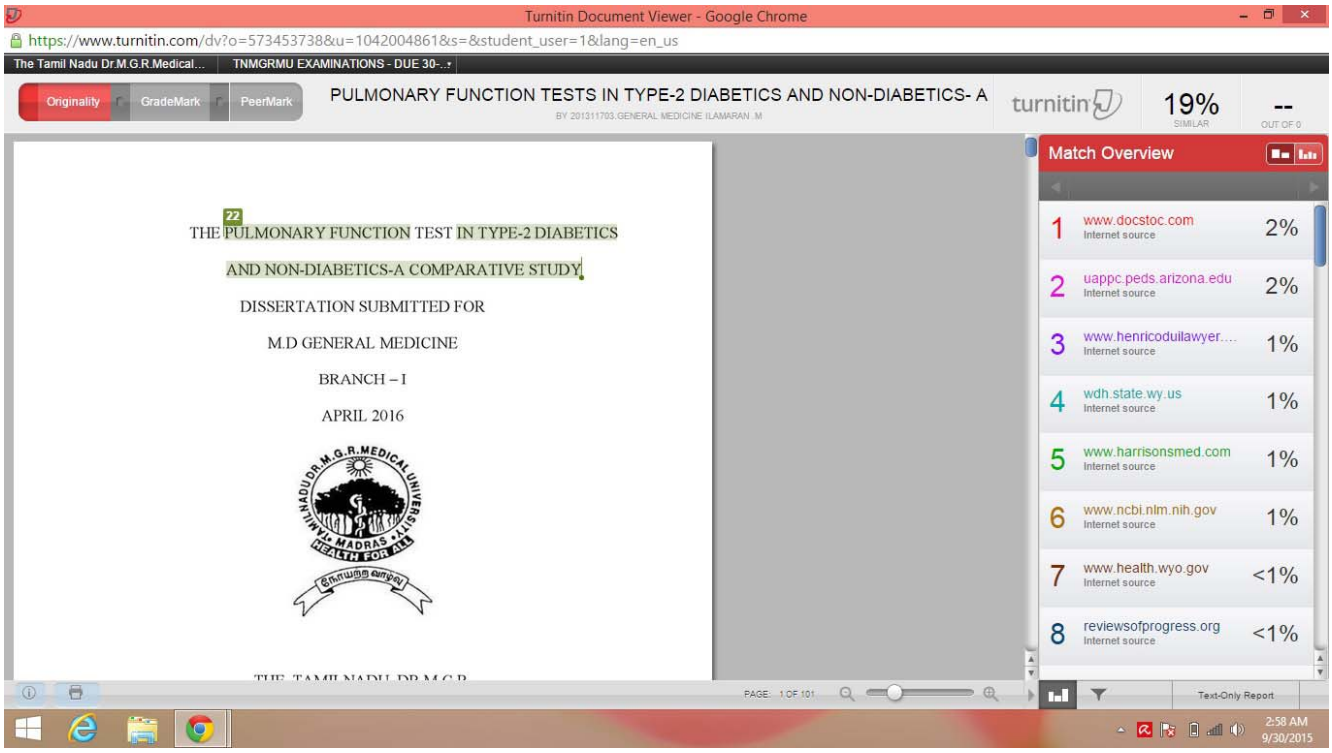
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